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Fertility and Labor Market Responses to Reductions in Mortality

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Non-Technical Summary

Rapid growth in women's labor force participation was one of the most remarkable economic changes of the 20th century in developed countries. In the United States, married women's labor supply grew from 10% to 25% between 1930 and 1950, and increased at a steady rate until 1990, when it reached 75% (Goldin, 2006). The initial increase has been attributed to increases in high school completion and the growth of office-based woman-friendly jobs, while the later increase has been associated with advances in home technology among other things (Goldin 2006). Our research suggests child mortality decline as a new contributor to the historic trend, and one that is likely to be pertinent to understanding demographic and economic change in developing countries today.

Our key idea is that declines in child mortality reduce both the number of children that women desire and the number of children they need to conceive in order to reach their desired number. This allows them to delay the initiation of fertility and to either join or remain in the labor market. On the intensive margin, this is reinforced by concomitant declines in child morbidity which reduce the time that women, the main caregivers, spend looking after sick children.

In sub-Saharan Africa in 2017, average female labor force participation was 63%, but this average masks significant heterogeneity, with participation ranging from 31% in Mauritania to 83% in Mozambique (World Bank). Consistent with our hypothesis, countries with lower child mortality tend to have higher female labor force participation. At the same time they have lower fertility as expected but, in contrast to the predictions of existing economic theory, higher childlessness. We propose a behavioural model which, by integrating fertility timing and labour force participation decisions of women, can explain this result. We test the predictions of this model on early 20th century American data.

To investigate our hypothesis, we leverage exogenous and sharp variation in child mortality created by the introduction of the first antibiotics in the US in 1937. These were sulfonamide (sulfa) drugs and they changed the standard of modern medicine. They treated major bacterial diseases including pneumonia, the leading cause of child mortality, and puerperal sepsis, a leading cause of maternal mortality. A "sulfa craze" followed, with 10% of the US population treated annually by 1941. Mortality rates from pneumonia – which was predominantly a disease of childhood – declined in the United States by 17-32% with the advent of sulfa drugs.

We find that women reduced their fertility when child survival improved, consistent with popular growth theory. We document reductions along the extensive *and* intensive margins of fertility: women who had children had fewer overall, and more women remained childless. The effect on childlessness is potentially puzzling, as modern economic theory posits that improvements in child health will make it *more* - not less - attractive to have at least one child (Becker and Lewis 1973, Aaronson et al. 2014).

This seeming paradox is resolved by incorporating into the decision-making framework the other choices women make. When child survival improves, women can delay fertility because there is less of a need to start having children early in life, since each pregnancy is more likely to succeed. With this increase in disposable time, women can enter or remain in the labor market. Being in the labor force opens the door to experience, for example, women may do well at work or develop a taste for work and either of these factors may modify their preferences. Alternatively, biological factors may limit fertility if it is delayed too long. These circumstances can result in eventual childlessness, even if this was not the woman's initial intention. Indeed, delay has been proposed as an explanation of the recent rise in childlessness in European countries like Italy (Kohler et al. 2017).

Consistent with the proposed mechanism, we find that after the decline in child mortality caused by the introduction of the first antibiotics, women had children later, were more likely to be in the labor market, achieved better occupations, worked longer hours, and were less likely to have ever married.

Looking also at the decline in maternal mortality that occurred alongside the decline in child mortality in late 1930s America, we find that maternal mortality decline encouraged higher fertility and thus had opposing effects on labour market outcomes of women who, at the time of the decline, had completed their education.

The burden of child mortality, maternal mortality and fertility the US in the 1930s was similar to that in many modern-day developing countries. Fertility remains high at 4.7 births per woman on average in Africa (United Nations 2015). Worldwide, pneumonia continues to be the leading infectious cause of death among children, accounting for 6 million under-5 deaths every year (Liu et al. 2016). Each day 830 women die in or around childbirth (Ceschia and Horton 2016). While eighty years have elapsed since the invention of antibiotics, the average consumption of antibiotics in West Africa is approximately 90% lower than in the United States, suggesting poor access (Hogberg et al. 2014).

Our findings imply that public investments in child mortality decline may liberate women from child bearing and rearing into economic activity. Female labor force participation has been argued to encourage long run economic growth (Bloom, Canning, Fink and Finlay, 2009). The economic empowerment of women has also been associated with greater investments in children (Baranov et al. 2017) and lower domestic violence (Aizer 2010). However, the extent to which this potential is realized will depend upon women being able to access appropriate employment. This in turn is a function of the nature of technological change in the economy (Goldin 2013), and of social norms which in many parts of the world still constrain women from availing of outside opportunities (Pande et al. 2017).

Our research also suggests that when maternal mortality declines, women who have already completed their education will tend to have additional children and reduce their labor market attachment. However, previous work suggests that women who are of school-going age will increase their investments in education and have greater labour force attachment.

Fertility and Labor Market Responses to Reductions in Mortality*

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Abstract

We investigate women's fertility, labor, and marriage market responses to large declines in child mortality in the U.S. Fertility declined on the intensive margin as expected. However, despite the increasing value of having at least one child, a larger share of women remained childless. We explain these findings with a new theory of fertility that includes fertility timing and labor force participation as choices. Consistent with the model's predictions, we find that reductions in child mortality led women to delay childbearing, increase their labor force participation, improve their occupational status, and to be less likely to have ever married.

Keywords: women's labor force participation, fertility timing, childlessness, child mortality, maternal mortality, medical innovation

JEL Classification: J13, I18

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1 Introduction

This paper investigates fertility, labor and marriage market responses to sharp declines in mortality and morbidity occasioned by a medical innovation. A vast literature has attempted to estimate how child mortality influences fertility and investments in children (e.g. Galor 2012, Soares 2005, Aaronson, Lange, and Mazumder 2014, Baudin, de la Croix, and Gobbi 2019, Kalemli-Ozcan 2003). We depart from these previous studies in two substantive ways. Most of these studies are benchmarked on the Becker and Lewis (1973) model of child production in which families simultaneously choose the quantity (fertility) and quality of children (investments). Our main contribution is that we allow for a third margin, namely fertility timing, and in this way we incorporate women's labor force participation decisions. Our insight is that child mortality decline increases the disposable time of women, allowing them to delay fertility and remain in or join the labor market. It is premised on the notion that with lower child mortality, women need to have fewer pregnancies in order to achieve their target number of children. This allows them to initiate childbearing later.[1] This makes it more attractive to invest in the labor market, and unnecessary to marry early. Thereafter, positive shocks to wage earnings, learning about the benefits of work, declines in fecundity with age, or indeed inertia, can lead to childlessness.[2] We outline a simple model capturing these features that can explain our empirical findings. Ours is the first attempt to analyse, within a single framework, how mortality may modify the timing of birth, childlessness, labor force participation and marriage.[3]

Our second contribution is that we provide the first analysis that distinguishes extensive from intensive margin fertility responses to declines in mortality and morbidity. Baudin, de la Croix, and Gobbi (2015) highlight that extensive and intensive margin fertility may or may not move in the same direction. They propose a structural model that can explain observed fertility patterns, but they focus on education and marriage. Aaronson, Lange, and Mazumder (2014), in another recent contribution, put forward the case for a distinction between extensive and intensive margin fertility choices with a Beckerian model, and they analyse responses to a school building intervention. In this literature, innovations in schooling and child health are similarly conceptualized, both bringing

[1] Four forces are likely to be at play here. First, the direct effect of more births surviving will lower replacement behavior in fertility (Bhalotra and van Soest (2008) estimate a replacement rate of 0.37). Second, it will tend to reduce hoarding behaviour that arises because mortality is uncertain. Third, because declines in mortality are typically associated with declines in morbidity (and thus an improved child health endowment), there is an increase in the incentive to invest in child quality, which will tend to reduce the target (desired) number of children. Fourth, women in this era spent a significant share of their time caring for sick children and, on average, an episode of pneumonia in a child under 15 led to 39 days of disability in the pre-antibiotic era (Britten (1942)). Thus a decline in sickness rates of surviving children will also have released the time of women for labor market engagement.

[2] This is not implausible. To take one related example from the literature, Acemoglu, Autor, and Lyle (2004) find that the aggregate demand shock that drew many women into the labor force during the WWII mobilization years was shortlived, having reversed itself by 1947. However, women continued to work in greater numbers after 1947. They rationalize this as follows: "...presumably because employment during the war changed their preferences, opportunities, and information about available work." (p. 501).

[3] Demographers have shown that birth intervals tend to be shorter when child mortality is high, and this is incorporated into a dynamic structural model by Bhalotra and van Soest (2008). However, this insight has not been linked to labor force participation, nor to childlessness.

about changes in the price of child quality. We argue that there is an important distinction between the two that has, hitherto, been neglected. Improvements in school availability only change the relative prices of child quality and quantity, leading families to move along the curve that traces the trade-off between these choices. However, improvements in child health may additionally influence fertility timing, changing the woman's allocation of time to effect a trade-off between fertility and her labor force participation. While both trade-offs (quantity vs quality of children and a woman's fertility vs her labor force participation) have been extensively studied in the literature, no previous work has incorporated discussion of the second trade-off in the analysis of the first. We discuss in more detail below the new insights provided by our distinguishing extensive and intensive margin fertility in response to mortality decline.

Our main findings are as follows. Sharp declines in child mortality are associated with women delaying childbearing and having fewer children. Fertility reduction is observed on the extensive *and* intensive margins: fewer women had three or more children, and a larger share remained childless. Importantly, we also observe that women were more likely to work, had higher occupational scores (a measure of the skill intensity of employment), worked longer hours and were less likely to have ever married.

The key finding that child mortality decline encourages fertility delay and higher rates of women's labor force participation augments research on the interplay between labor market, marriage and fertility choices (Adda, Dustmann, and Stevens 2017, Jensen 2012, Goldin and Katz 2002, Goldin 1997), and on fertility timing (de la Croix and Pommeret 2018, Herr 2016, Choi 2017, Ananat and Hungerman 2012). It provides a new angle on understanding the trade-off between career and extensive margin fertility decisions (see e.g. Lundborg, Plug, and Rasmussen (2017) in another context).

Previous work has linked fertility delay to marriage and labor market incentives (Caucutt, Guner, and Knowles 2002), but not to falling child mortality. A related literature has documented the liberating influences of the expansion of women's education and the introduction of the birth control pill, which enabled fertility delay, later marriage, and labor force participation (Goldin and Katz 2002, Goldin 2006, Bailey 2006); we show that child mortality decline had similar effects.[4]

Although the transition to low fertility is central in theories of economic growth (Galor and Weil 1996), the drivers of the fertility transition remain hotly debated (Galor 2012). In a seminal paper, Aaronson, Lange, and Mazumder (2014) propose that distinguishing extensive and intensive margin fertility responses permits discrimination between competing theories of the demographic transition. Their main insight is that, in the Becker and Lewis (1973) quantity-quality model of fertility, child quality and child quantity are substitutes on the intensive margin, but complements on the extensive margin. In particular, their model implies that when changes in the opportunity

[4]The literature on the pill is thus a good reference point for our analysis, where fertility delay is a mechanism. The contraceptive pill was a technology that revolutionized women's control over their fertility and the literature has documented that it led to later fertility and higher labor market participation. However, in contrast to our findings, the pill had no impact on completed fertility at either margin (Ananat and Hungerman 2012). A possible explanation for this difference is that women in the antibiotic era were more likely to have to choose between career and family, while women in the pill era could more easily have both (Goldin 2004, Coles and Francesconi 2018).

cost of women's time are the dominant driver, the two margins will move together. In contrast, when changes in the price of child quality (proxied by a school building program) are the dominant driver, the two margins will move in opposite directions.[5]

However, we find that a change in the price of child quality (proxied by child mortality and morbidity decline) leads to the two margins moving in the same direction.[6] We propose that an explanation of this difference is that changes in the price of child quality directly influence the opportunity cost of women's time. Essentially, we show that our findings are consistent with the predictions of a model that extends the work of Aaronson, Lange, and Mazumder (2014) and Becker and Lewis (1973) to allow fertility to be a dynamic choice variable determined jointly with labor force participation. This innovation unites the two forces often posited as competing explanations of the demographic transition: increases in the opportunity cost of women's time and improvements in child survival.

It also generates a new theory of childlessness that contributes to a recently active literature in this area (Baudin, de la Croix, and Gobbi 2019, Baudin, de la Croix, and Gobbi 2015, Gobbi 2013, Currie and Schwandt 2014, Ananat, Gruber, and Levine 2007) [7] Of particular interest, Baudin, de la Croix, and Gobbi (2015) argue that the opportunity cost of work (indicated by higher education) is a driver of extensive margin fertility, while we emphasize that shifts in child mortality may create a feedback channel running from fertility to work. Finally, it produces a new prediction, drawing a causal link between child mortality decline and increases in women's labor force participation. To the extent that marriage is a prerequisite for fertility, this also generates the prediction that child mortality decline will reduce the probability of marriage.

There were dramatic increases in women's labor force participation in the mid-20th century: for example, married women's labor force participation more than doubled, from 10% to 25%, between 1930-1950 (Goldin 2006). This occurred alongside sharp falls in child mortality, and such changes are afoot in many developing countries today. Increases in women's labor force participation in this era have been associated with the high school movement and the emergence of more woman-friendly jobs (Goldin 2006), but we propose child mortality decline as another channel. Our findings are in

[5] Aaronson, Lange, and Mazumder (2014) demonstrate that their hypothesis holds in the data. They use the variation created by the Rosenwald school building program targeted at the African-American population in the American South in the early 20th century. The program generated a decrease in the price of child quality for women who were of reproductive age when it was introduced, and an increase in the opportunity cost of time for the next generation of women who acquired more education on account of the program. First-generation women who *faced a decrease in the price of child quality* for their potential births were more likely to have at least one child, but had fewer children conditional on at least one. Thus, consistent with their predictions, the extensive and intensive margin responses are in opposite directions. Moreover, they show that for the children of these women (who received more education and hence had a *higher opportunity cost of time*), the extensive and intensive margin responses are in the same direction, both being lower. Overall, their findings for an increase in the opportunity cost of time resemble our findings for a decrease in the price of child quality due to improvements in child health and survival.

[6] Our empirical estimates show that while the intensive margin response to child mortality decline is similar to the intensive margin response to school building, the extensive margin responses act in opposite directions.

[7] Recent models of childlessness suggest that childlessness at the upper end of the education distribution is voluntary, being driven by the opportunity cost of childbearing (Baudin, de la Croix, and Gobbi 2015, also see Aaronson, Lange, and Mazumder 2014). Baudin, de la Croix, and Gobbi (2015) discuss involuntary childlessness at the bottom of the education distribution as arising from poverty. Ours is a fundamentally different proposition, namely that declines in child mortality can lead to childlessness via fertility delay - some part of which is arguably involuntary.

line with previous work arguing that the cohorts of women in our sample were those that typically had to trade off career and family (Goldin 2004, Aaronson et al. 2017).

The rest of this introduction discusses the identification strategy, the magnitudes of the estimated effects, a series of robustness checks, and the external validity and current day relevance of our findings. For identification of causal effects, we leverage the antibiotic revolution of 1937 in the United States. The first antibiotics, sulfonamide drugs, led to sharp drops in both mortality and morbidity from pneumonia, the leading cause of child mortality (Jayachandran, Lleras-Muney, and Smith 2010). This effectively reduced the price of investing in children (i.e. in child quality) by improving child health and thus the returns to other investments in children. Previous work has demonstrated this by showing significant improvements in education, employment and income among cohorts born just after the arrival of antibiotics (Bhalotra and Venkataramani 2012).[8]

We exploit the trend break in pneumonia mortality in 1937, together with the fact that states with higher pre-intervention levels of these mortality rates experienced larger declines in mortality in the post-antibiotic era. This difference-in-difference strategy follows Acemoglu and Johnson (2007), Bleakley (2007) and Bhalotra and Venkataramani (2012). Using the United States decennial population censuses for 1940-1970, we identify women of reproductive age in a window around the introduction of antibiotics in 1937 and study whether their fertility timing is influenced by plausibly exogenous declines in child mortality. We then find the same birth cohorts of women when they have completed (or progressed) their fertility and estimate models for the total number of children, distinguishing between the extensive and intensive margins of fertility, and estimating the impact on the entire distribution of fertility so as to understand where along the intensive margin reductions in fertility occur. We use a similar estimation strategy to analyze impacts of mortality decline on labor and marriage market outcomes.

Sulfa drugs were only effective for certain antibacterial infections - thus, they were effective for pneumonia, but not for tuberculosis. We control for mortality rates from tuberculosis and four other "placebo" causes of death for which the drugs were not effective. This helps undermine the competing hypothesis that we capture not pneumonia-driven child mortality decline but rather generic health improvements. We additionally control for other sulfa-treatable causes of death. Among these, the only highly prevalent cause of death was maternal mortality. Puerperal sepsis, a postpartum infection of the reproductive tract, was a leading cause of maternal mortality at this time and was treatable with sulfa drugs (Thomasson and Treber 2008, Albanesi and Olivetti 2014, Albanesi and Olivetti 2016).

We show that there was sufficient variation in the geographic distribution of pneumonia and maternal mortality, allowing us to identify their impacts independently. Previous studies pursuing impacts of child mortality decline on fertility have typically paid no attention to the possibility that the intervention leading to child mortality decline may have simultaneously led to maternal

[8] We focus on the *generation of women who were of childbearing age* when sulfa drugs arrived, and were making choices over their fertility and labor market lifecycle profiles. In contrast, Bhalotra and Venkataramani (2012) focus on the generation who were *children when sulfa drugs arrived*, showing the human capital impact on individuals who had access to antibiotics when they were young.

mortality decline. This was the case in the antibiotic era, and will tend to be the case for other therapeutic innovations in infectious disease control, and for policy changes such as the introduction of reproductive and child health programs (Bhalotra, Karlsson, and Nilsson 2017). It is important to distinguish a woman's responses to her own mortality risk at birth from the risk of child mortality because they can have opposing (and possibly offsetting) effects on fertility and labor supply.

Indeed, we find that the sulfa-led maternal mortality decline, by reducing the opportunity cost of childbearing, had opposing effects to child mortality decline: it led to higher fertility and lower labor force participation, although these effects are sensitive to specification and to sample. There is limited evidence on the size or nature of fertility responses to maternal mortality decline, with the important exception of Albanesi and Olivetti (2014, 2016), who also document a similar result but take a more macroeconomic approach.

Our results may appear to stand at odds with recent work arguing that improvements in women's general health lead to increases in productivity and hence in women's labor force participation, with reductions in fertility following from this (Bloom, Kuhn, and Prettner 2015). We focus on a particular facet of women's health - maternal mortality associated with childbirth, which acts to make fertility less costly and thereby reduces women's labor force participation. Thus, while there is no contradiction between the two results, it is notable that improvements in women's reproductive health may have opposite effects on fertility and labor force participation to improvements in women's general health.[9]

We now elaborate on the magnitudes of the estimated effects. We use a decline from the 75th to the 25th percentile of the pre-intervention pneumonia mortality distribution to scale our estimates. Using a hazard specification on the sample of women of reproductive age in a short window around the arrival of the antibiotics and leveraging the discrete change in their availability in the potential birth year, we find that pneumonia mortality decline led to a 0.6 percentage point (6.9%) reduction in the annual probability of birth, and a 0.3 percentage point (5.9%) reduction in the annual probability of becoming a mother (extensive margin).

Using an alternative specification that models the number of children ever born to the same cohorts of women observed later in the lifecourse, we estimate that the same reduction in pneumonia mortality led to a 4.6 percentage point increase in the probability of childlessness for women who had not yet completed their fertility at the time of census enumeration (12.8% of baseline), and to 0.18 fewer (net) children conditional on at least one (7% of the baseline mean of 2.61). By the time women completed their fertility, the impact on childlessness is reduced by two thirds, indicating significant delay in the childbearing sample.

In line with the predictions of our theoretical model, we find evidence that can explain the extensive margin fertility response with reference to labor market participation. We estimate an increase in the probability of women's labor force participation of 2.6 percentage points (7%), an

[9] Our sample cohorts were women of reproductive age who had largely completed their education. Maternal mortality decline can, by increasing life expectancy and increasing returns to education, lead to larger investments in education and thereby to higher labor force participation. This is relevant for cohorts who are still acquiring education when maternal mortality declines, as in Jayachandran and Lleras-Muney (2009).

increase of 6.6 percentage points in occupational score, and more hours worked (1.15 hours more per week). Importantly, we find that child mortality decline increased the joint probability that a woman was both childless and in the labor force by 13% of the baseline probability (20.5%), and this increase was driven entirely by a decline in the number of women who were stay-at-home mothers.[10] The estimated impacts on marriage rates are smaller, with the chances of being ever-married declining by 1.5 percentage points (1.7%).

Turning to the estimated impacts of maternal mortality decline, we find that the average woman in the childbearing sample was 3.3 percentage points (9% of baseline) less likely to be childless. However, this effect is estimated to be zero for the sample of women with completed fertility, suggesting that the decline in maternal mortality altered the timing of births but not the desired number. Maternal mortality decline was associated with a decline in the probability of labor force participation of 3.8 percentage and in working hours of 1.08 hours, while the probability that a woman had ever married increased by 1.9 percentage points (2.3%). These results are consistent with the earlier timing of birth.

The impacts of child and maternal mortality are not comparable because they are on different scales, the inter-quartile range being of different magnitudes in the two pre-antibiotic distributions. Moreover, the compliers responding to declines in pneumonia and maternal mortality are likely different (see Section 6.5). Overall, to the extent that different types of women are at the margin for responses to child and maternal mortality, this suggests a possible polarization among women, with some increasing labor supply and decreasing their fertility, and others doing the opposite.

We demonstrate the robustness of our findings to several coherence and specification checks. We present evidence that women had access to fertility control in this early era, so that they could alter the timing of their births. We also provide evidence that fertility often deviates from plan, this being relevant to the emergence of childlessness following deferral of fertility. We investigate and reject differential trends in outcomes between states with higher versus lower disease burdens in the pre-sulfa era. In all specifications, we control for placebo diseases (diseases not treatable with sulfa drugs), measures of health care access and socioeconomic conditions at the state-year level. Following Pei, Pischke, and Schwandt (2018), we also conduct a balance test using these controls. To allow for unobservable omitted trends, we condition on region-year fixed effects and, in a variant, on state-specific trends. We further account for variability across the states and over time in the quality of birth and death statistics.

We show that our findings are not driven by potentially confounding events, such as the Second World War and its evidenced impacts on women's labor force participation (Acemoglu, Autor, and Lyle 2004, Goldin and Olivetti 2013), New Deal spending and the Dust Bowl, and we account for mean reversion, the introduction of prescription charges, measurement error in the measurement of pneumonia mortality, survivorship bias, sample selection and endogenous migration. We show comparable estimates using gross and net (i.e. net of survival) measures of fertility to address

[10] This relationship is also evident in the summary statistics. 56% of childless women were employed compared to 24% of mothers, and 51% of childless women were ever-married compared with 99% of mothers.

potential limitations of each measure from the census data. We also address potential concerns about the measurement of fertility being sensitive to children leaving home, or to child mortality.

Given the evidence discussed earlier that women's health influences women's labor force participation (Bloom, Kuhn, and Prettner 2015), we tested whether child mortality decline in our data was proxying for omitted improvements in women's health occasioned by the antibiotics. In fact, improvements in mortality rates from pneumonia among adults were statistically negligible (see Figures 4 and 6), but we nevertheless allow for separate impacts of reductions in pneumonia among adults and among children. We find that women's labor force participation responded to improvements in child but not adult pneumonia.

We confirm that the relationships between child mortality decline and childlessness, total fertility and women's labor force participation that we identify are also evident as stylized facts in historical cross-state data for the United States, and in contemporary cross-country data. This underlines the broad scope of our findings.

These findings are of potential relevance today. Pneumonia is the leading cause of death among children today, and puerperal sepsis continues to be a major cause of maternal mortality. Child and maternal mortality rates in developing countries remain high: 6 million under-5 children continue to die every year, while maternal mortality stands at around 800 deaths each day (Liu et al. 2016). Fertility also remains high, at 4.7 births per woman in Africa, for example (United Nations 2015). While eighty years have elapsed since the innovation of antibiotics, the average consumption of antibiotics in West Africa is approximately 90% lower than in the United States, despite much higher rates of infectious disease, marking poor access (Hogberg et al. 2014).

Our findings suggest that a benefit of policies that reduce child mortality is that they can "liberate" women from early childbearing and multiple pregnancies and the disempowerment that often accompanies this practice. However, the size of these knock-on effects will depend upon the availability of labor market opportunities for women (Coles and Francesconi 2018).

The remainder of the paper proceeds as follows. Section 2 explains the context and documents the decline in child and maternal mortality rates occasioned by the sulfa drug revolution. Section 3 outlines a theoretical framework that guides our analysis of childlessness and women's labor force participation. Section 4 describes the empirical strategy and Section 5 the data. Section 6 discusses the estimated effects of sulfa exposure on fertility, labor market outcomes and marriage market outcomes, and an extensive set of robustness checks. Section 7 presents evidence for the external validity of our findings, and Section 8 concludes.

2 Contextual Information

2.1 Mortality Rates and the Sulfa Drug Revolution

The United States in the 20th century was characterized by high levels of maternal and child mortality (Britten 1942, Linder and Grove 1947). The arrival of the first antibiotics - sulphonamides, or sulfa drugs - drastically altered the standard of medical care, creating large and sharp changes

in morbidity and mortality from a number of bacterial infections. Sulfa drugs were discovered by German chemists experimenting with textile dyes in 1932 and then became available in the United States starting in 1937, achieving wide penetration in the consumer pharmaceuticals market (Lesch 2007). This was enabled by low costs (especially for a life-saving drug), the lack of prescription requirements to purchase the drugs (which were established only in 1939), and the lack of a binding patent.[11] By all accounts, there was a "sulfa craze", with adoption being widespread and quick (Jayachandran, Lleras-Muney, and Smith 2010, Lerner 1991).

Sulfa drugs were particularly effective in treating pneumonia among children and puerperal sepsis among new mothers, both of which were previously managed by supportive care (Thomasson and Treber 2008, Jayachandran, Lleras-Muney, and Smith 2010).[12] Pneumonia was the leading infectious cause of child morbidity and mortality, and the third leading cause overall (after death from premature birth and congenital defects). Pneumonia accounted for 17% of infant deaths in the United States in the 1930s. Mortality rates from pneumonia are U-shaped in age. Child mortality from pneumonia (under-5s) in the United States stood at an average of 11.8 per 1000 population between 1930-36, of which the majority occurred among infants (under-1s; 8.2 per 1000); the adult rate for the same period was 0.4, while the elderly rate (above-65s) was 3.5 (see Figure 2). The higher exposure of infants to pneumonia, together with the greater plasticity of their development, imply that impacts of pneumonia decline are likely to have been most important for individuals exposed to the decline in their infancy. A marker of the substantive importance of pneumonia mortality decline among children born in the sulfa era is that reduced exposure to pneumonia in infancy had large and statistically significant impacts on their educational attainment, employment, income, and disability when adults (Bhalotra and Venkataramani 2012).

In addition to reducing mortality, the arrival of sulfa drugs led to significant reductions in morbidity (Greengard et al. 1943, Hodes et al. 1939, Moody and Knouf 1940). Prior to the introduction of antibiotics, pneumonia was a debilitating disease with typical spells often lasting 39 days and children tending to have recurrent spells (Britten 1942). With the arrival of sulfa drugs, the severity and length of these episodes decreased (Connolly et al. 2012), and this was documented in clinical trials among hospital inpatients (Greengard et al. 1943, Moody and Knouf 1940). Relevant to the ensuing discussion, a reduction in pneumonia morbidity among children will have increased the disposable time of women, who were the main care-givers for children and the sick.

Puerperal sepsis - an ascending bacterial infection of the reproductive tract that can occur soon after birth - was a leading contributor to the high maternal mortality rates that characterized the

[11] The chemical structure of sulfonamide had previously been documented as part of a PhD thesis in the early 1900s. By the time it was established in the 1930s that it had antibacterial properties, the patent had expired and anyone was allowed to produce sulfonamide for commercial purposes.

[12] Sulfa drugs also reduced mortality and morbidity from skin and soft tissue infections and meningitis (Jayachandran, Lleras-Muney, and Smith 2010). However, the incidence of these diseases was very low and they made insignificant contributions to both infant and maternal mortality. For example, the all-age mortality rate from skin diseases in the U.S. was 1.8 per 1000 in 1930, and from meningitis 2.1, compared to an all-cause infant mortality rate of 69 (Vital Statistics).

pre-antibiotic era. This condition accounted for 40% of the 6.4 deaths per 1000 live births in 1930 (Vital Statistics). Maternal mortality was the tip of an iceberg, the mass of which represented high rates of maternal morbidity (Albanesi and Olivetti 2014). Sulphonamides were instrumental in reducing both mortality and morbidity from puerperal sepsis and thus improving the safety of childbirth.

2.2 Fertility Control and Planning

As we model changes in fertility timing, it is important to discuss the extent to which women in this era were able to control their fertility. There is considerable evidence that women born in the early 20th century were able and willing to practise fertility control (Morgan 1991). The early 20th century in the U.S. featured the birth control movement, led by political radicals Emma Goldman, Mary Dennett and Margaret Sanger, who argued the importance of birth control, particularly among low income women who were burdened by more children than they desired.[13] Before the arrival of the birth control pill in the 1960s, couples used diaphragms, latex condoms, vaginal suppositories, withdrawal and douching techniques (Engelman 2011), with the invention of the diaphragm in 1882 being particularly crucial to the advent of effective fertility control by women.[14,15]

Relevant to our prediction that childlessness may involuntarily follow from delayed fertility and labor market participation, there is evidence that, despite the availability of contraception, women often mis-forecast their fertility. A survey of women entering college in 1976 showed that most accurately predicted their future working lives but while 82% expected to have had a child by age 37, only 69% did (Goldin 2006). The idea that delaying fertility can lead to childlessness is integral to popular explanations for lowest-low fertility in Europe, although the processes they suggest are different from ours, being unrelated to child mortality (Kohler, Billari, and Ortega 2006).

2.3 Women's Labor Force Participation and Marriage

In the 1930s, there was a substantial increase in female labor force participation, with an increase of 15.5 percentage points, from 10% to 25%, on the extensive margin among married women (Goldin 2006).[16] This was further encouraged by a virtual elimination of marriage bars by the early 1940s.[17]

[13] Margaret Sanger was particularly active. She motivated the opening of the first birth control clinic in 1916, which was shut down, followed by a clinic in 1923, which was not shut down. These clinics were the precursor to Planned Parenthood and Sanger is considered the founder of the modern birth control movement.

[14] The relationship between fertility control and career choices has been discussed in sociology, for example see Wilkie (1981), Hayford (2013), Lundquist, Budig, Curtis, and Teachman (2009), Bloom and Trussell (1984). In particular, see Murray and Lagger (2001), who analyzes this in the context of the United States demographic transition in the 19th century.

[15] As we shall see, our results imply fertility control. If we had only observed an increase in childlessness in response to pneumonia mortality decline, this could be attributed to changes in fecundity or marriage rates, but we additionally observe a reduction in higher parity births, which is evidence of fertility control.

[16] Figure A.1 in Appendix B depicts the long-run trends in labor force participation, fertility and marriage rates during this period.

[17] Marriage bars were regulations that prevented married women from working - see Goldin (2006).

In the estimation sample that we describe below, with married and unmarried women drawn from the 1940-1970 censuses, the average labor force participation rate was 37%, with 34% of women in the 1940 census reporting being in the labor force. Previous work has argued that important drivers of the increase in labor force participation in this period included higher rates of high school completion, the arrival of "nice" jobs, such as secretarial work in offices that reduced the stigma associated with married women working, and the virtual elimination of marriage bars by the early 1940s. Regardless of education, most working women were engaged in such typing-oriented jobs, and teaching (Goldin 2006). We provide the first evidence that child mortality decline may have contributed to this rise in women's labor force participation.

In our sample, 85% of women were ever-married and the average age at marriage was 21. Marriage and fertility choices were closely related in this period, with only 8.5% of births being out of wedlock (Bachu 1999).[18]

3 A Model of Fertility and Labor Market Choices

In this section, we outline a stylized theoretical model that extends the quantity-quality framework of fertility of Becker and Lewis (1973) and Aaronson, Lange, and Mazumder (2014). The purpose of the model is essentially to formalize the intuition of our empirical findings. In the classical model (outlined in Appendix D), parents choose the quality and quantity of children, subject to prices and income. Aaronson, Lange, and Mazumder (2014) separate extensive from intensive margin responses and make the important observation that while quantity and quality tend to be substitutes on the intensive margin, they must be complementary on the extensive margin. This implies that when the price of child quality declines, childlessness falls. We extend the model further by introducing fertility timing as a third choice variable and thereby incorporating women's labor maket choices in the framework. We show that, under certain conditions, this reverses the predictions of the model for childlessness. It also generates new predictions for causal effects of child health and improvements in survival on women's labor market participation.

3.1 Dynamic fertility choices

Consider a woman whose decisions about her fertility and income-earning activity evolve over time, as a function of child mortality risk and labor market shocks. The woman is fertile at dates $t = 1, ..., T$. Each period she has one chance to get pregnant, and the woman always attempts to have a child until she has her target number of children. Pregnancy leads to a surviving child with probability $1 - \lambda$, where λ is the child mortality rate. The woman can work up to the date t_0 of

[18] In 1936, a year before the introduction of sulfa drugs, Popenoe (1936) conducted a survey among students at the University of Southern California asking them to describe the history of all permanently childless couples that they knew. This shows that 22% of couples were believed to be childless due to the wife's career, and 16% childless due to economic pressure, suggesting that fertility and labor market choices were linked during this period, even for married women. Although the survey oversampled wealthier and more educated women, it gives some indication of possible reasons for childlessness during the 1930s among married women.

her first pregnancy, consistent with the "job then family" lifecycle pattern of the cohorts of women living in the sulfa drug era (Goldin 2004). If she never gets pregnant, she can work up to T.[19] The woman's initial wages are y. Each period she has the potential to get promoted with probability p, and in this event her wages rise from y to $Y > y$; for simplicity, she can only be promoted once. This mechanism (hereafter "promotion") can capture any positive labor market shock, such as positive shocks to job satisfaction or development of tastes for work. We will show that this can result in childlessness if a woman decides not to get pregnant after this favorable shock. Fertility delay motivated by potential benefits on the labor market is similarly a feature of the model in Goldin and Katz (2002), where the birth control pill serves as an effective technology for women to delay fertility and marriage and invest in their skills.

The woman's lifetime income is

$$I = \sum_{t=1}^{t_0-1} \tilde{y}_t,$$

where $\tilde{y}_t \in \{y, Y\}$ denotes the realized wage each period. At time T, when fertility is completed and the number n of children is known, the woman chooses the quality e of her children and her consumption c, to solve the problem

$$\max_{c,e} U(c, n, e) \text{ subject to } n(\tau^q + \tau^e e) + c \leq I,$$

where τ^q is the price of quantity, and τ^e is the per-child price of quality. This quantity-quality trade-off follows the canonical model in Becker and Lewis (1973) and Galor (2012). Let $e^\star(n;\tau)$ be the optimal choice of quality when the woman has had n children and prices are $\tau = (\tau^e, \tau^q)$. Substituting the binding budget constraint yields the woman's maximized utility, conditional on having n children, as

$$\sum_{t=1}^{t_0} \tilde{y}_t + V(n;\tau),$$

where

$$V(n;\tau) = u\left(n, e^\star(n;\tau)\right) - n\left(\tau^q + \tau^e e^\star(n;\tau)\right)$$

is the indirect utility from having n children when prices are τ. We can show that the value $V(n;\tau)$ of having $n \geq 1$ children is strictly *decreasing* in the prices of quantity and quality. Thus, the predictions of Aaronson, Lange, and Mazumder (2014) are nested within our model. Moreover, holding constant the timing of pregnancy, it is clear that a decrease in child mortality (an increase in λ) mechanically increases the number of successful pregnancies, and therefore also reduces the number of childless women.

The introduction of sulfa drugs reduced child mortality and morbidity, lowering the price of child quality and quantity. The Aaronson, Lange, and Mazumder (2014) model would lead us to expect less childlessness. It would also lead us to expect lower intensive margin fertility, if the

[19] To save notation, we do not allow women to work after their fertile period, but this assumption does not affect the direction of the results.

reduction in the price of quality dominates the reduction in the price of quantity.[20]

Incorporating fertility timing can reverse the predicted effect of declines in child mortality on childlessness. To see this, consider the simplest case where the woman is fertile for $T = 2$ periods. We focus on the extensive margin by assuming that $u(1, e) > u(n, e)$ for all $n > 1$. Under this condition, it is optimal to have at most one child. We write $V(\tau) = V(1, \tau)$ for simplicity. We further normalize the pre-promotion wage to $y = 0$. To solve the model, it is useful to separate three parametric cases. First, if $V(\tau) < 0$, it is never optimal to get pregnant, and the woman always remains childless by choice. Second, if $Y < (1 - \lambda)V(\tau)$, it is never optimal to enter the labor market, because even promotion cannot lead to an outcome that dominates getting pregnant. We focus on the third case - the intermediate region with $0 < V(\tau) < Y/(1 - \lambda)$, where a woman trades off work against starting a family.

3.2 The trade-off between early and late pregnancy

In the intermediate parametric region, it is optimal to remain in the labor market, and thus to remain childless, if promotion occurs at $t = 2$. The key trade-off is between (i) getting pregnant at $t = 1$ and giving up the possibility of promotion (which was the reality in the sulfa drug era in the US - see Goldin 2004), and (ii) delaying fertility, entering the labor market for one period and waiting until $t = 2$ to attempt childbearing if promotion does not occur, which entails the risk of ending up with no surviving children.[21]

It pays to delay pregnancy and enter the labor market at $t = 1$, instead of getting pregnant early, if and only if the net value of waiting, $N(\tau, \lambda)$, is greater than zero, where

$$\begin{aligned} N(\tau, \lambda) &= [pY + (1 - p)V(\tau)(1 - \lambda)] - [V(\tau)(1 - \lambda^2)] \\ &= \underbrace{p\,[Y - V(\tau)(1 - \lambda)]}_{\text{option value of delay}} - \underbrace{V(\tau)\lambda(1 - \lambda)}_{\text{insurance value of early pregnancy}}. \end{aligned} \tag{1}$$

The option value of delay measures the expected utility gain from getting promoted. It is increasing in λ: the option of working for high wages is more valuable, relative to attempting a pregnancy, if child mortality is high. The insurance value of early pregnancy measures the expected utility gain from having a second chance: with probability λ, the first pregnancy does not survive, but the second survives with probability $1 - \lambda$. The second chance thus adds value V with probability $\lambda(1 - \lambda)$. The insurance value is a hump-shaped function of λ with its peak at $\lambda = 1/2$. Intuitively, when $\lambda \simeq 0$, the first pregnancy almost never fails, so the insurance value is low. When $\lambda \simeq 1$, both pregnancies almost always fail, so again the insurance value is low. The insurance value is therefore highest for intermediate levels of λ.

[20] In general, there is also an income effect, but it is usual in the literature to assume quasilinear utility, so that the substitution effect dominates.

[21] de la Croix and Pommeret (2018) and Pestieau and Ponthiere (2014, 2015) also consider the question of fertility timing; the former examines the role of income risk, while the latter take a lifecycle approach in a macroeconomic model.

Other things equal, the incentive to delay fertility and participate in the labor market increases with child mortality decline if the partial derivative of $N(\tau, \lambda)$ with respect to λ is positive:

$$\frac{\partial N(\tau, \lambda)}{\partial \lambda} = V(\tau)\left(1 - 2\lambda - p\right) > 0. \tag{2}$$

This occurs when

$$\lambda < \frac{1-p}{2}. \tag{3}$$

A decrease in child mortality encourages fertility delay and labor force participation if child mortality $\lambda < \frac{1}{2}$ and the probability p of getting promoted is low enough. How can we interpret this? Suppose that λ falls. Recall that the insurance value of early pregnancy, $V(\tau)\lambda(1-\lambda)$, is a hump-shaped function of λ with a maximum at $\lambda = \frac{1}{2}$. If $\lambda > \frac{1}{2}$, then the insurance value of getting pregnant increases with a marginal fall in child mortality, which discourages delay. In contrast, when $\lambda < \frac{1}{2}$, then the insurance value declines in response to this marginal fall in λ, which encourages delay. Second, the option value of delay, $p[Y - V(1-\lambda)]$, declines by pV when λ declines. If p is small enough, then the decline in the option value of delay, which reduces the net value of delay $N(\lambda, \tau)$, is small. A decline in the insurance value increases the net value of delay. Thus, if p is small, then on balance the net value of delay tends to increase in response to the decline in λ. A population average child mortality rate of 47 per 1000 live births in 1939 (Dowell, Kupronis, Zell, and Shay 2000) implies that $\lambda = \frac{47}{1000}$, and the condition is satisfied if the probability of promotion is less than 90.6%, which seems highly plausible.[22]

3.3 Population effects of decreased child mortality

To obtain empirical predictions for the overall effect of child mortality decline on fertility and labor market participation, incorporating the effect of changes in the prices of child quality and quantity, fertility delay and mechanical effects on child survival, we derive the population impact of a decline in λ, allowing prices to adjust as well. Suppose there is a population of women $i \in [0, 1]$ who for simplicity have a constant probability of promotion p, and of which a fraction $\eta(\tau)$ never wish to get pregnant (i.e. $V^i(\tau) < 0$) and a fraction $\delta(\lambda, \tau)$ prefer to delay (i.e. $N^i(\tau, \lambda) > 0$).[23] The fraction of women who never wish to get pregnant does not directly depend on the child survival rate λ, because the value $V^i(\tau)$ of having one surviving child is fully determined by the prices τ

[22] Women in this era primarily worked in teaching and typing jobs, where the probability of promotion was low (Goldin 2004).

[23] If Y^i and $V^i(\tau)$ are measurable functions of i, then we can write the fraction of women who never wish to get pregnant is

$$\eta(\tau) = \int_{i:V^i(\tau)<0} d\mu,$$

and the fraction of women who wish to delay as

$$\delta(\lambda, \tau) = \int_{i:N^i(\tau,\lambda)>0} d\mu.$$

of child quality and quantity. The proportion who wish to delay does depend on λ, because the probability of child survival affects the trade-off between early and late pregnancy.

Recall that women can have zero children or one child in the model. Integrating over the population, the proportion of women who have one child is

$$C(\lambda, \tau) = \delta(\lambda, \tau)(1-p)(1-\lambda) + (1 - \delta(\lambda, \tau) - \eta(\tau))(1-\lambda^2).$$

The following Proposition summarizes the impact of a decline in child mortality on the number of childless women in the population:

Proposition 1 *The total population effect of a child mortality shock (a decline in λ) on fertility is $-\frac{dC(\lambda,\tau)}{d\lambda}$. This can be decomposed into the direct effect of λ and the indirect effect through prices τ:*

$$\underbrace{-\frac{dC(\lambda,\tau)}{d\lambda}}_{total\ effect} = \underbrace{-\frac{\partial C(\lambda,\tau)}{\partial \lambda}}_{direct\ effect} + \underbrace{\frac{\partial C(\lambda,\tau)}{\partial \tau} \cdot \left(-\frac{d\tau}{d\lambda}\right)}_{price\ effect}.$$

The price effect or quantity-quality tradeoff effect (the second term) is always positive: the decline in the prices of child quantity and quality reduces the number of childless women in the population. The direct effect (the first term) can be decomposed into a mechanical effect (lower childlessness due to higher survival rates), which is always positive, and a dynamic effect (higher childlessness due to more women delaying and potentially being exposed to a positive labor market shock, leaving them childless):

$$\underbrace{-\frac{\partial C(\lambda,\tau)}{\partial \lambda}}_{direct\ effect} = \underbrace{2\lambda(1 - \delta(\lambda,\tau) - \eta(\tau)) + (1-p)\delta(\lambda,\tau)}_{mechanical\ effect} - \underbrace{(1-\lambda)(p+\lambda)\left[-\frac{d\delta(\lambda,\tau)}{d\lambda}\right]}_{dynamic\ effect}.$$

The dynamic effect has the same sign as the individual woman's incentives in condition (2), so that childlessness can increase with child mortality decline if and only if

$$\lambda < \frac{1-p}{2}. \tag{4}$$

Proof. In Appendix D. ■

Thus, childlessness can increase in the population when child mortality declines under the same condition that governs whether fertility delay and labor market participation increase. Figure 1 shows the population effect of a decrease in λ on fertility delay when Equation (4) holds. The solid line is given by Equation (4) and delineates the two groups of women: those above the line prefer to delay fertility and enter the labor market, while those below the line prefer to start childbearing in the first period.

When child mortality falls, the solid line shifts down to the dotted line. More women prefer to delay instead of childbearing in the first period. Switchers are those women who are close to

indifferent between earning income Y and enjoying utility from childbearing V. The shaded area to the left of the y-axis depicts women with $V < 0$, who always prefer to work instead of having a child.

Note that the classical quality-quantity trade-off, and the effect of sulfa drugs through prices τ, can also be understood intuitively in terms of Figure 1. If sulfa drugs cause a decrease in τ^q and/or τ^e, the value V^i of having a child increases for each woman, and the distribution of V in the population shifts to the right (in the sense of first-order stochastic dominance). Therefore, a group of women move from the "never pregnant" to the "delay" region, and another group of women move from "delay" to "rush". Both groups are now less likely to remain childless which, in the absence of any changes in fertility timing or labor market choices, translates to an overall decrease in childlessness in the population.

3.4 Summary of Empirical Predictions

Extending the classical quantity-quality model to allow for fertility delay and labor market participation yields the following distinct empirical predictions:

- In response to a fall in child mortality, there will be an increase in the number of women delaying their first birth and participating in the labor market if the *dynamic effect* dominates the *price effect* and the *mechanical effect* (condition (4)).
- If the dynamic effect dominates, we will also see an increase in the number of women remaining childless.
- There may be negative impacts on marriage probabilities, if fertility and marriage are linked.

We shall see that the evidence lines up with the dynamic channel of fertility delay illustrated in the model.

3.5 Extensions of the Model

Other Shocks Learning about the benefits of future work, changes in fertility preferences, and changes in the biological ability to have children with age (fecundity) have predictions that are similar to the effect of promotion; see Appendix D, where we also discuss how predictions change if the probability of promotion p is not constant.

Intensive Margin We have focused our analysis on the extensive margin fertility response. On the intensive margin, reductions in the price of child quality that dominate reductions in the price of child quantity will reduce the desired number of children, n^*. This would provide an additional reason to delay fertility since lowering n^* reduces the insurance value of early pregnancy - because fewer successful pregnancies are needed to achieve a lower target number of children.

Education Decisions Our model abstracts away from education decisions because we study decisions of women of childbearing age, most of whom had completed their education.[24] In Appendix D, we analyze heterogeneous responses by education. Childlessness in our model may include an involuntary component but women purposively make lifecycle decisions about labor force participation and childbearing based on their preferences over children and their returns on the labor market, and switchers (i.e. women whose choices are affected by a marginal decline in mortality) can theoretically occur at any point of the education distribution. In particular, switchers are women who are close to indifferent between being in the labor market and starting a family. Shocks can arise from promotion prospects but also from other sources like preferences and fecundity. As discussed earlier, this contrasts with Baudin, de la Croix, and Gobbi (2015) and Aaronson, Lange, and Mazumder (2014), where voluntary childlessness occurs at the top end of the education distribution due to a high opportunity cost of childbearing and, in Baudin, de la Croix, and Gobbi (2015), childlessness at the low end of the education distribution occurs due to a lack of resources needed to afford children, which can be interpreted as being involuntary.

Maternal Mortality We have focused on understanding the impact of child mortality decline on fertility and labor market outcomes. The impact of maternal mortality decline can also be understood in this framework. Maternal mortality decline reduces the cost of having a child. This can be interpreted as a decline in the price of child quantity, τ^n. The model predicts higher fertility on both the extensive and intensive margins, less fertility delay, and lower labor market participation.[25]

4 Research Strategy

4.1 Identifying Variation in Mortality Rates

When introduced, sulfonamide (sulfa) drugs were available nationwide, at pharmacies, and at an affordable cost (Lesch 2007). The timing of the introduction of antibiotics created sharp variation across cohorts in exposure to disease. Figure 3 shows trend breaks in pneumonia mortality in 1937. The steepest post-sulfa decline in pneumonia mortality was amongst infants and young children (Figure 4). Moreover, there was considerable geographic dispersion in the pre-intervention levels

[24] In Section 6.3 we report that, using the sample of women who had not completed their education by 1937, we find that exposure to sulfa-led declines in child (pneumonia) mortality was associated with an increase in high school completion rates, while maternal mortality decline had the opposite effect. Our findings are consistent with Goldin, Katz, and Kuziemko (2006) who discuss the timing of the expansion of college education. Note that this occurred later, among *children* of the sulfa cohorts.

[25] Maternal mortality may also have direct impacts on labor supply (i.e. not just through fertility choices). In particular, maternal mortality decline tends to be associated with declining morbidities around childbirth and a longer life expectancy, which may lead to higher labor market participation. Thus, there are two potentially competing channels. This channel is unlikely to operate in our setting since we look at outcomes for women who were of reproductive age and had largely completed their education when the sharp drop in maternal mortality occurred. Nevertheless, we impose no restriction in the estimation, and the empirical results will be able to discriminate between the competing hypotheses.

of pneumonia mortality, and we exploit the fact that states initially most burdened by pneumonia experienced the largest declines in these mortality rates following the introduction of sulfa drugs; see Figure A.2 for maps showing the geographic dispersion of pneumonia mortality. Figure 5 shows the post-sulfa convergence in pneumonia mortality rates across the U.S. states. This convergence was most marked for children and infants (Figure 6). In the cited figures we also show trend breaks, geographic dispersion and convergence for maternal mortality which, as discussed, was treatable with sulfa drugs and we consistently control for.[26] We exploit the large variation across U.S. states in pre-intervention levels of the disease burden and the post-1937 decline in mortality rates in a difference-in-difference approach. Our identification strategy is similar to that in Acemoglu and Johnson (2007), Bleakley (2007) and Bhalotra and Venkataramani (2012).[27] Following a tradition in the literature (Almond 2006, Bozzoli, Deaton, and Quintana-Domeque 2009), we assume that changes in mortality are a proxy for both mortality and morbidity, so that mortality decline also captures improvements in the health of survivors.

4.2 Estimating Equations

We use two complementary modelling approaches. First, we identify the impact of mortality decline on birth timing by estimating the probability of birth of a woman in a given year in 1930-1943 as a function of the discrete change in the availability of sulfa drugs. Second, so as to estimate effects on childlessness and total fertility, we find the same birth cohorts of women in later census files and estimate the "stock" of children as a function of exposure to sulfa drugs, defined as the number of a woman's fertile years in the post-sulfa period. In both cases, we interact the relevant measure of sulfa exposure with pre-sulfa mortality rates at the birth state level. We adopt a similar strategy to the second approach for identification of impacts on labor market and marriage outcomes.

Hazard Model: Birth Timing We use the sample of women of reproductive age during 1930-43 and model the hazard of birth in this short window around 1937 using a data file expanded to the woman-year level to create a within-woman panel of potential birth years. The estimating equation is:

$$\begin{aligned}\Pr(Y_{jst} = 1) &= \beta + \beta_1 * prePneumonia_s * post1937_t \\ &+ \beta_2 * preMMR_s * post1937_t \\ &+ \boldsymbol{\beta_3}\mathbf{hazardcontrols}_j + \boldsymbol{\beta_4}\mathbf{womanbirthyear}_j + \boldsymbol{\beta_5}\mathbf{race}_j + \boldsymbol{\beta_6}\mathbf{education}_j \\ &+ \boldsymbol{\beta_7}\boldsymbol{\lambda}_s + \boldsymbol{\beta_8}\boldsymbol{\theta}_t + \boldsymbol{\beta_9}\boldsymbol{\psi}_{t,r} + \boldsymbol{\beta}_{10} * post1937_t * \mathbf{statecontrols}_s + u_{jst},\end{aligned} \tag{5}$$

[26] The patterns in the figures are formalized with tests of significance in Tables A.4 and A.5: pneumonia mortality declined on average 9.6% per year from 1937, while maternal mortality declined 12.7% per year. This per-year decline in pneumonia mortality was driven entirely by the infant and child rate, while the adult and elderly rates exhibited a modest annual *increase* between 1937 and 1943. The estimated convergence coefficients for pneumonia and maternal mortality are -0.29 and -0.22 respectively, implying that an interquartile shift in pneumonia mortality (-0.26) due to the arrival of sulfa drugs was associated with a 7.5% reduction in pneumonia mortality, while an interquartile shift in maternal mortality (-1.84) was associated with a 40.5% reduction in maternal mortality.

[27] These are amongst the small set of studies that attempt to account for the endogeneity of mortality.

where Y_{jst} is a binary indicator that equals 1 if woman j born in state s gave birth in year t, $post1937_t$ equals one if the potential birth year of the child is 1937 or after and $prePneumonia_s$ is the pre-1937 state-level pneumonia mortality rate, constructed as an average over 1930-1936. We control for maternal mortality (MMR), using the same formulation as used for pneumonia mortality, allowing a trend break in the series in 1937 and leveraging observed post-sulfa convergence across states. Although pre-intervention levels of pneumonia and maternal mortality are positively correlated, they also exhibit independent variation across the U.S. states (Figure A.3). In assigning pre-intervention mortality rates and state-level controls to women, we use their birth state. This is because migration decisions after birth are potentially endogenous. As a result, we may underestimate treatment effects for women who moved into areas with the largest health gains. However, as a check, we re-estimated the equation assigning all state-level variables to the resident state of the woman at the time of census enumeration, and the results were qualitatively similar - see Section 6.6, where we also discuss modelling migration as an outcome and find no evidence of endogenous migration.

The coefficient of interest is β_1, which captures the causal effect of pneumonia mortality decline on the probability of birth. The parameter β_2 will reflect causal effects of maternal mortality decline on the birth probability. The vector $\mathbf{hazardcontrols}_j$ consists of indicator variables for years since last birth with the count starting at age 15 and restarting after every birth, and the birth order of the next birth. We include fixed effects for the woman's race ($\mathbf{race}_j$), education ($\mathbf{education}_j$), birth state ($\boldsymbol{\lambda}_s$), birth year ($\mathbf{womanbirthyear}_j$), and the calendar year of the potential birth ($\boldsymbol{\theta}_t$). The calendar year fixed effects control for year-specific events, such as the recession of 1937-38 (notice that child mortality fell from this year and onwards, despite the recession). Education will proxy (imperfectly) for potential wages and fertility preferences.

We also investigate controls for woman fixed effects, which will capture any unobservables that determine compliance and are also potentially correlated with fertility preferences (see Appendix F). We estimate equation (5) as a logistic regression, yielding estimates for a discrete time proportional hazard model. Standard errors are clustered at the birth state level to account for serial correlation in outcomes within states (Bertrand, Duflo, and Mullainathan 2004).

Threats to inference arise if state-level convergence in outcomes would have occurred even without the arrival of sulfa drugs, or if there are unobservables that correlate with baseline mortality rates that predict different trends in outcomes.[28] We address this by controlling for a rich vector of relevant state health and socioeconomic characteristics (detailed below). Importantly, we include the pre-intervention level of these variables interacted with the indicator for birth years 1937 and onwards ($post1937_t * \mathbf{statecontrols}_s$). As this is the formulation used for pneumonia mortality, it subjects our interpretation of the coefficients of interest to a strong test of whether they may spuriously capture the impacts of post-1937 changes in other baseline state characteristics. To further allow for unobservable trends, we include census region-year fixed effects ($\boldsymbol{\psi}_{t,r}$).

[28] Candidate omitted variables include pre-intervention trends in other factors that influence fertility, such as income or skill-biased technological change that differentially increased the returns to quality (i.e. human capital investment) or that produced increased opportunities for women.

In order to distinguish between the extensive and intensive margins of fertility timing, we extend this analysis to estimate the impacts of mortality decline on the probability of a first birth and, separately, the probability of higher order births. In particular, we estimate equation (5) on the subset of the sample where we only include woman-year observations in which the woman would be at risk of giving birth to her first child (the extensive margin), and only including observations where the woman would be giving birth to her second or higher-order child (the intensive margin).

Stock Model: Total Fertility and Childlessness Any changes in the timing of births may be adjusted for later in the fertility cycle. The contraceptive pill, for example, led women to delay fertility but there was no impact on completed fertility (Ananat and Hungerman 2012). In order to assess whether there was compensating variation in fertility in later years, we estimate models for the stock of births.

For this, we use a sample of women of the same birth cohorts as in the hazard sample, namely women of reproductive age around the time of the introduction of sulfa drugs, but observed in later census years. First, we identify women who are at least 40 at the time of census enumeration and have thus plausibly completed fertility. Second, to illuminate the role of fertility delay, we use an alternative sample that includes all women aged 18-40 at census enumeration. Results of this model of women of childbearing age bridge the birth timing estimates with the completed fertility estimates.[29]

We model exposure as the number of fertile years spent in the post-sulfa era, interacting this with pre-sulfa mortality rates. Using the stock model, we give up the ability to identify responses from a discontinuity in exposure during the fertile years, but we gain an estimate of whether fertility responses simply reflect the advancing or deferral of overall fertility. The estimating equation is:

$$\begin{aligned} B_{jsc} &= \alpha + \alpha_1 * prePneumonia_s * sulfayears_j \\ &\quad + \alpha_2 * preMMR_s * sulfayears_j \\ &\quad + \boldsymbol{\alpha}_3 * \mathbf{birthyear}_c + \boldsymbol{\alpha}_4 \mathbf{race}_j + \boldsymbol{\alpha}_5 \mathbf{education}_j + \boldsymbol{\alpha}_6 \boldsymbol{\lambda}_s \\ &\quad + \boldsymbol{\alpha}_7 * sulfayears_j * \mathbf{statecontrols}_s + e_{jsc}, \end{aligned} \tag{6}$$

where B_{jsc} denotes the total number of births to woman j born in state s in cohort c as recorded at the time of the census, and $sulfayears_j$ is the number of fertile years of woman j during which she is exposed to sulfa drugs. We assume women are fertile between the ages of 15 and 40. When we use the sample of women aged 18 to 40, $sulfayears_j$ may include future years: for example, a woman aged 33 at the time of the 1950 census who was 20 in 1937, would have 20 fertile sulfa years in total, of which 7 are in the future. If women make dynamic fertility choices, they care about

[29] In particular, the oldest woman in the stock sample at the time of antibiotics introduction was 44 in 1937, so that she was reproductive until 1933, and the youngest woman was six in 1937, so that she was reproductive from 1946 onwards; see Section 5 for details.

their total exposure to sulfa drugs, and not only their past exposure.[30]

Controls are similar to those in the hazard model, including fixed effects for the woman's birth cohort and birth state, her race and education, and a vector of state-specific variables interacted with $sulfayears_j$, to create a formulation that is parallel to the variables of interest. Standard errors are clustered at the state level. To identify the extensive margin response, we redefine the dependent variable as a binary indicator for non-zero versus zero births. To identify the intensive margin response, we re-estimate equation (6) restricting the sample to women with at least one birth.

Labor and Marriage Market Outcomes We model labor and marriage market outcomes as in the stock model specification, by varying the dependent variable:

$$\begin{aligned} L_{jsc} &= \gamma + \gamma_1 * prePneumonia_s * sulfayears_j \\ &\quad +\gamma_2 * preMMR_s * sulfayears_j \\ &\quad +\boldsymbol{\gamma}_3 * \mathbf{birthyear}_c + \boldsymbol{\gamma}_4 \mathbf{race}_j + \boldsymbol{\gamma}_5 \mathbf{education}_j + \boldsymbol{\gamma}_6 \boldsymbol{\lambda}_s \\ &\quad +\boldsymbol{\gamma}_7 * sulfayears_j * \mathbf{statecontrols}_s + e_{jsc}, \end{aligned} \tag{7}$$

where L_{jsc} includes the following labor market outcomes: whether in the labor force; whether working; the hours worked in the past week; the Hauser-Warren occupational score (a measure of the skill intensity of employment; see Appendix A), and personal income in the last year. We estimate the impact on the following marriage market outcomes: whether currently married, ever married and the age at first marriage conditional on ever having married. We focus on women aged 18-50 at census, thus covering the sample of women of childbearing age (18-40) and those who have completed their fertility (40-50).

4.3 Potential Confounders and Control Variables

As discussed, since the independent variable of interest is an interaction term between the pre-sulfa pneumonia mortality rate and an indicator for post-sulfa birth cohorts, the main threat to inference is that there may be omitted state-level trends correlated with pre-sulfa disease burdens and outcomes, or with diffusion of sulfa drugs. To adjust for this possibility, we include a rich vector of state-specific variables in the same formulation as the variables of interest i.e. interacted with sulfa exposure. These are the logarithms of per capita state income, public health spending, education spending, and the numbers of schools, hospitals and physicians, women's labor force participation and women's literacy. Income per capita, for example, captures underlying convergence in economic development across states, which may affect mortality and fertility. It is noteworthy, however, that

[30] In Section 6.6 we discuss replacing the linearly evolving exposure measure with a binary measure that allows us to compare women who were fully exposed with women who were unexposed, removing women who were partially exposed.

income per capita explains only 10.7% of the variation in pneumonia mortality and 17.4% of the variation in maternal mortality in the pre-sulfa era, with a fixed effect for race explaining more (especially for maternal mortality). Further, we will show below that we find opposing effects of child and maternal mortality on fertility and labor market outcomes. If state convergence in economic development were a confounder of our findings, it would need to be correlated in opposite directions with convergence in child and maternal mortality, which is not the case in the raw data.

We control for mortality rates from five causes of death that were not influenced by sulfa drugs ("placebo diseases"). This ensures that we are not simply capturing the effects of secular changes in the overall disease environment. Including deaths from communicable diseases (tuberculosis, diarrhea, malaria) helps control for state-specific changes in, for instance, sanitation, public health programs and housing, which may have coincided with the arrival of sulfa drugs and influenced fertility decisions. Controlling for tuberculosis is especially useful as predictors of pneumonia are very similar to predictors of tuberculosis, so that we can isolate the variation stemming from anitbiotics by using the fact that pneumonia was, while tuberculosis was not, treatable with sulfa drugs. We also include non-communicable diseases (cancer, heart disease) to control for factors such as health care quality and access. For all of these variables, we use a pre-sulfa state-level measure, which is their average over 1930-1936, and interact this with our cohort-varying measure of exposure: $post1937_t$ in the hazard and $sulfayears_j$ in the stock model.[31]

The first half of the twentieth century was an era in which there was a staggered process by which the states were joining the U.S. Vital Statistics registration system, resulting in variation in the quality of birth and mortality data (Eriksson, Niemesh, and Thomasson 2017). To address this, we include the years that each state entered the U.S. Vital Statistics birth registration and death registration systems, interacted with the measure of exposure.

We further include census region-year fixed effects to control flexibly for underlying trends, though only in the hazard model, because we lack power to estimate the large number of census region-birth cohort fixed effects given the stock model sample size. We conduct a number of further checks on specification and data, highlighted in Section 1 and detailed in Section 6.6.

5 Data

Data on individual outcomes are taken from the United States Census Microdata (Ruggles et al. 2010). Appendix A describes the data used for the explanatory variables and provides more detail on how the outcome variables are defined.

Fertility: Hazard Data The hazard dataset is constructed from pooled census microdata using the 1940 and 1950 1% samples.[32] We restrict the analysis to births that occurred in a short window

[31] For female labor force participation and literacy, we use the value in 1930 as annual series are unavailable.

[32] In all datasets, we restrict the sample to US born women not residing in group quarters who are resident in their birth state at the time of the census. The latter limits bias that could arise due to migration; see Section 6.6 for robustness checks on migration.

around 1937, namely 1930-1943, to limit the role of confounding events, such as the influenza epidemic of 1928-9 and the increasingly widespread use of penicillin after 1943. In Section 6.6, we investigate sensitivity to variation in the analysis window. We select women who were of childbearing age (15-40) at any time between 1930 and 1943 and expand the data to create a woman-level panel, with observations for every year in which a woman was at risk of giving birth. Thus, we only include woman-year observations in which a woman was aged 15-40 in that year, so that we capture choices during the fertile period. The cohorts in the hazard sample were born in the years 1890-1928. Cohorts born between 1928-1931 did not become fertile until 1944, which falls outside the hazard sample window.

We use a measure of net fertility that derives from a record of the children living in the mother's household at the time of enumeration.[33] Using a variable that links child records to mothers, we constructed a complete history of live births for each woman, restricting births to biological births (95% of children in the household) that occurred in the U.S. As is standard, this measure excludes any pregnancies that did not result in live births, and any deaths that occurred after birth but before the census. It also excludes any children that had left home. The latest census we use is the 1950 census, where the oldest child born during the estimation period 1930-1943 would have been 20, which minimises concerns about missing older children. In Section 6.6 we motivate and discuss estimates that measure fertility using only children under the age of 10, following Aaronson, Lange, and Mazumder (2014).

Fertility: Stock Data The stock dataset is compiled from pooled census microdata containing the 1% samples of the 1940, 1950, 1960 and 1970 censuses.[34] The sample includes birth cohorts 1893-1931 who were age 6-44 in 1937. We exclude women aged five and under in 1937 as they were potentially directly treated by antibiotics as children. We include not only women aged 15-40 in the period 1930-1943 (as in the hazard sample) but also women who would have been exposed to the sulfa drug era throughout their reproductive years (aged 6-15 in 1937) and women who would not have been exposed at all (aged 40-44 in 1937). We find these women in different censuses, depending on the age at which we are interested in their fertility and labor market outcomes. Note that all women aged 15-40 in 1937 were partially treated. In Section 6.6 we discuss a specification with a binary measure of exposure, comparing fully exposed with unexposed women.

As in the hazard sample, we focus on net fertility (children resident in the household), but also estimate the impacts on gross fertility (number of live births) to show that both measures yield comparable estimates.[35] To measure completed fertility, we identify women aged 40-50 at the time of census enumeration. For example, the 1917 cohort will have turned 40 in 1957, so we find them

[33] In theories of population increase and economic growth, the focus is on the number of surviving children a woman has, or net fertility (Acemoglu and Johnson 2007, Brueckner and Schwandt 2015). Women looking to achieve a target number of births will also set the target in net terms (Galor 2012).

[34] Although the 1960 and later censuses were flat samples, the 1940 and 1950 censuses oversampled some groups. We re-estimated our main results with survey weights and they were very similar to the reported results in Section 6.

[35] The live births question was asked only to ever-married women in the 1940 and 1950 censuses (and all women subsequently). In our sample, 92% of women are ever-married in the 1940 and 1950 censuses. We have checked that our results are robust to considering only ever-married women in the 1960 and 1970 censuses.

in the 1960 census. This allows us to estimate whether any change in birth timing that we observe reflects intertemporal substitution, or a change in the overall number of children, and the upper age ceiling limits bias due to children moving out of the home. We also estimate the effect of sulfa exposure on fertility timing in the stock model, using a sample of childbearing women aged 18 to 40 at the time of census enumeration.[36,37]

Gross fertility will tend to overestimate total fertility and underestimate childlessness (as some women with a positive number of live births will end up childless due to children dying), while net fertility may underestimate fertility due to children moving out. As we estimate the effect of sulfa on both measures of completed fertility, we are able to redress the weaknesses of each.[38]

Labor Market and Marriage Data Data on labor market and marriage outcomes are from the same census files as the stock data on fertility. We consider women aged 18 to 50 at the time of the census, in order to reflect the same sample of women as for fertility outcomes.

Cause-specific Mortality Rates and Indicators of Economic Development Data on baseline rates of diseases were taken from several volumes of the U.S. Vital Statistics (Grove and Hetzel 1968, Linder and Grove 1947, Ruggles et al. 2010, Bureau 1943). For the pre-intervention levels of maternal and pneumonia mortality we use the state-level average over the years 1930-36 of the mortality rate. For maternal mortality, this is defined per 1000 live births and for pneumonia it is the all-age pneumonia-influenza mortality rate per 1000 population. We use an all-age rate in place of the child rate in order to reduce measurement error: infant deaths were known to be under-reported during this time (Linder and Grove 1947, Eriksson, Niemesh, and Thomasson 2017), with under-reporting varying by state, making the child rate noisier than the all-age rate. However, the overwhelming source of variation in the all-age rate was child and infant mortality, with the all-age decline after 1937 driven by the steep decline in child deaths from pneumonia - see also the discussion in Section 2.1 and Figures 4 and 6. Thus, we opt to use the all-age pneumonia mortality rate to measure the decline in child mortality. However, in Section 6.6, we show that our estimates are robust to using the all-age rate as an instrumental variable for the child rate, and that they are robust, though attenuated, when using the child mortality rate directly, which is consistent with under-reporting in the child rate. Later, to address the specific concern that adult mortality rates may be driving the labor force participation result, we introduce the adult rate in the model and

[36] Given that we are restricted to observing younger women, the cohorts in this sample were born between 1900-1931: women born between 1893-1900 would have been at least 40 by the time of the 1940 census, and hence have completed their fertility.

[37] We have intentionally opted to select samples based on age at census enumeration, in order to compare outcomes during childbearing and after childbearing years. This means that there is a small difference between the cohorts analyzed across the childbearing and completed fertility samples. We have verified that our main results (fertility, labor and marriage market) are very similar when we restrict the sample of each estimate to have exactly the same cohorts.

[38] A threat to inference with net fertility arises if the age at which children leave home is correlated with state level baseline pneumonia or maternal mortality. Although this should be absorbed by state fixed effects, the results are similar when considering net fertility among different age groups at census, focusing on younger women where children are less likely to have moved out; see Appendix F.

show that the coefficients on this are not statistically different from zero.

We use the mortality rate for pneumonia and influenza rather than the mortality rate for pneumonia alone, also to reduce measurement error. The diseases shared symptoms, such as cough and fever, and were difficult to distinguish in the 1930s. Moreover, they were intrinsically related since pneumonia commonly followed from an initial influenza infection.[39]

We collated data on under-2 diarrhea, heart disease, cancer, malaria, and tuberculosis mortality from U.S. Vital Statistics as control variables, creating state-specific and cause-specific pre-sulfa era mortality rates between 1930 and 1936. We compiled time series data on the socioeconomic and infrastructure variables described in Section 4.3 from several sources: see Appendix A.

Summary Statistics Tables A.1 and A.2 provide descriptive statistics for the hazard and stock samples, as well as for the state-specific disease environment measures and controls. In the hazard model, the woman-year observations are balanced before and after the intervention, with an annual mean probability of birth of 8.7%. All-age pneumonia and influenza mortality before the intervention was on average 1.09 per 1000 population, while maternal mortality was on average 6.26 per 1000 live births. In the stock model sample, using completed (gross) fertility, the average woman was exposed to sulfa for 14.9 years, 19% of women were childless at the end of their reproductive years, average total fertility was 2.6 (unconditional on at least one birth) and 3.2 conditional on at least one. Focusing on net fertility among women of childbearing age, the same figures are 20 years, 36% childless, 1.7 births unconditional, and 2.6 conditional on at least one. The mean age at first birth in the stock sample was 24.1, and 26.7 at second birth. In the labor market sample, 37% of women were in the labor force, and 35% were working, with average working hours of 13 per week.[40] 73% of women reported being currently married, and 85% had been married at least once previous to the date of census enumeration.

6 Results

6.1 Hazard Model: Birth Timing

We find that the conditional probability of birth declined following sulfa-led reductions in pneumonia mortality (Table 1).[41] This result is consistent with the standard theory of the quantity-quality tradeoff: the decline in pneumonia mortality and morbidity generated sharp improvements in the

[39] In fact, annual time series data are only available for the combined measure, reflecting the problem of isolating pneumonia from influenza deaths. However, using decadal data that provide separate series for pneumonia and influenza, we note that (a) pneumonia dominated the combined mortality rate, with 8.9 deaths per 1000 in 1930, compared to 1.3 deaths per 1000 live births from influenza, and (b) the decline in the combined mortality rate between 1930 and 1940 was entirely on account of a decline in the pneumonia mortality rate: while influenza rates fluctuated considerably with epidemics and seasons, the average influenza rate was steady across the decade.

[40] This includes women reporting zero hours of work; focusing only on positive working hours, the average was 37.9.

[41] Columns (1)-(3) in Table 1 pool a woman's births, and in so doing assume independence across recurrent events. Standard errors are clustered at the state level, which allows for non-independence within mothers as, by construction, they do not move state. The extensive margin result in Column (4) is robust to this concern, as the extensive margin has one event (the first birth).

child health endowment and in child survival, both of which will have motivated reductions in fertility.[42] The coefficient on pneumonia mortality is robust to the series of controls that we described earlier. Columns (4) and (5) in Table 1 show results for the extensive and intensive margin responses. Notably, fertility declined on both margins in response to pneumonia mortality decline.

An interquartile shift in (baseline) pneumonia mortality (-0.26) implies a 0.6 percentage point reduction in the annual probability of birth, which relative to the annual mean of 8.6% before 1937 is a reduction of 6.9% (column 3). The estimates in column (4) for the extensive margin response imply a 0.3 percentage point reduction in the annual probability of transitioning to motherhood after 1937, which is 5.9% of the pre-intervention mean of 5.1%. For a woman exposed to sulfa drugs for ten years of her fertile period, this is a 3 percentage point increase in the probability of being childless in the same period. The reduction in the annual probability of transitioning into motherhood shows fertility delay (i.e. an increase in the time to first birth). This is consistent with our theoretical framework linking child mortality decline with fertility delay.

The intensive margin result shows that the same interquartile shift implies a 0.25 percentage point reduction in the probability of transitioning to a higher order birth after 1937, which is 1.7% of the pre-intervention mean of 14.9%. Thus, the decline in pneumonia mortality delayed the transition to first and higher order births. The delay to the first birth was about three times the delay to higher order births, which is important for the childlessness result that we document below, using the stock measure of fertility.[43]

We also display the coefficient on maternal mortality, as this was a sulfa-treatable cause of death. Our expectation was that this will have made birth less costly for women and hence more attractive, other things equal. In line with this, the coefficient on maternal mortality is positive at the extensive and intensive margins. It is statistically significant with basic controls and census region-year fixed effects, but is sensitive to the inclusion of controls for state-year variables. Although both coefficients are positive, there is no statistically significant impact of the reduction in maternal mortality on birth probability on the extensive or intensive margins.

In Appendix F, columns (2) and (3) in Table A.32, we show that the coefficient on pneumonia mortality is not significantly smaller when maternal mortality is dropped from the model, which alleviates concern that the correlation in the geographic distribution of pre-1937 pneumonia and maternal mortality rates may bias the coefficient of interest.

Event Study We estimated an event study specification for probability of birth similar to column (3) of Table 1, with the difference that rather than interact exposure to pneumonia mortality with the indicator *post*1937, we interact it with indicators for every year in the sample period (1930-

[42] The increase in the child survival rate will have also caused a decline in the per-child price of quantity; however, the overall decline in fertility in response to child mortality decline suggests that the decline in the price of quality dominated, causing a reduction in desired fertility (see also the model in Section 3).

[43] Standardizing the logistic coefficients by the variance in the outcome variable yields coefficients that are directly comparable across samples, and shows that the negative effect of the reduction in pneumonia mortality on the extensive margin birth probability in column (4) is similar to the effect on the overall birth probability in column (3), and both are approximately three times the size of the effect on the intensive margin in column (5).

1943, with 1937 as the base year). The resulting coefficients are plotted in Figure 7. While this exercise is challenged by statistical power and not all coefficients are statistically significant, the plot suggests a discrete change in 1937. These plots also provide a test of the identifying assumption that pre-trends in birth outcomes in states with high versus low pre-intervention disease burdens were not different insofar as the coefficients show no trend before 1937. The figures suggest that our results are unlikely to be driven by underlying trends in economic conditions, as convergence in economic conditions was gradual, while we find sharp changes in birth probability after 1937. We conduct further checks on pre-trends in Section 6.6 below. Overall, our findings show that women delayed childbearing in response to improvements in child survival and child health.

Figure 7 also shows an event study for maternal mortality. As in Table 1, fertility responses to maternal mortality are positive. Although the coefficients in the Table were not robust to state-year varying controls, the figure shows a clear tendency for a break in the series from 1937.

6.2 Stock Model: Childlessness and Total Fertility

The hazard model describes how the flow of births changed in response to the introduction of sulfa drugs. To assess whether the response was only an intertemporal substitution or whether there was a change in total fertility, we estimate the effect of the intervention on the stock of births. We start by "bridging" estimates of fertility measured as a birth stock for women who are still childbearing. These estimates will capture a combination of fertility delay and changes in fertility targets. We then report results for women who have completed childbearing.

6.2.1 Fertility Among Women of Childbearing Age

As in the hazard model, we use information on children living with the mother to construct measures of the stock of children. The sample contains women aged 18-40 at the time of the census and aged 6-44 in 1937, identified by their birth cohort in the 1940-1970 censuses.[44]

The estimates indicate that the reduction in pneumonia mortality had a significant, negative effect on fertility that is robust to inclusion of the controls described earlier and that is significant at both the intensive and extensive margins (columns (1)-(3), Table 2). Sensitivity to controls is reported in Table A.13 in Appendix F.

The specification in column (1) implies that an interquartile decline in pneumonia mortality (0.26) was associated with 0.013 fewer births for an additional year of exposure to sulfa drugs. Relative to the mean of 1.66 children in the estimating sample, a woman with the average years of exposure in this sample (20) had 0.25 fewer births, or 15% of baseline.[45] Conditional upon having at least one birth, women had 0.18 fewer births for mean levels of exposure (which is 7% of the

[44] For some of these women, a disproportionate share of whom are younger and hence sulfa-treated during their reproductive years, fertility is right-truncated. This will be captured by fixed effects for the woman's age, which we consistently include.

[45] We use means in the estimating sample to calculate % baseline numbers throughout the stock model results because some outcomes were not recorded in the 1930 (pre-sulfa) census. This means that we will underestimate the % baseline effect sizes.

conditional mean of 2.6 births). A similar calculation implies a 0.23 percentage point increase in the probability of childlessness for an additional year of sulfa exposure, and a 4.6 percentage point increase in the probability of childlessness for the mean duration of exposure (13% of the baseline rate of childlessness, which is 36% in this childbearing sample). The coefficients also suggest that the reduction in maternal mortality led women to have more children, but these coefficients are only stable and significant at the extensive margin.[46]

Impacts on the Distribution of Fertility An increase in childlessness will mechanically decrease intensive margin fertility. For instance, if women who would otherwise have had one child switch to having none, then the increase in childlessness will be mirrored in a decrease in the share of women with one child. So as to identify where the intensive margin adjustment occurs, we estimate sulfa-led changes in the distribution of fertility. We define indicator variables for the number of children being 0, 1, 2, 3 and 4+ and estimate equation (6) separately for each of these outcomes. Figures 8 (a) and (b) plot the coefficients on the pneumonia and maternal mortality exposure terms. We see a leftward shift of the fertility distribution in response to reductions in pneumonia mortality, with statistically significant responses at the two ends of the distribution: in response to pneumonia (child) mortality decline, women were more likely to be childless and less likely to have three or more children. Although the coefficients are negative, there was no significant change in the share of women with either one or two children.

In contrast, we see that the reduction in maternal mortality resulted in a rightward shift of the fertility distribution. Although only the coefficient on childlessness is statistically significant, the other coefficients indicate that families of zero or one children became less common, while families of two or more became more common.

6.2.2 Completed Fertility

We now discuss estimates of equation (6) on a sample of women who had completed their fertility at the time of census enumeration. This allows us to identify impacts on total fertility, purging any effects of delay, or intertemporal substitution. We first present results using the net fertility measure, which relies on counts of children living at home, for which we impose an upper limit of age at census of 50 to reduce the omission of children who have left home.[47] To check that our results are similar when using an alternative measure of fertility that does not rely on observing children living at home, we estimate the impact of mortality decline on gross fertility, which is

[46] Although these coefficients are not precisely estimated, an interquartile decline in maternal mortality is estimated to have resulted in 0.12 more births overall, and 0.03 more births on the intensive margin. The impact on total fertility is smaller than estimated in Albanesi and Olivetti (2014), who find that a decline in maternal mortality of 1 death per 1000 live births (roughly half of the 1.84 drop we use) is associated with a rise in completed fertility of 0.27 children per married woman (roughly double that of the 0.12 increase that we estimate). They use a different sample of women and a different estimation approach, but our results point in the same direction.

[47] We will underestimate fertility counts because some of the older children will have left home . Since older women in the sample will have had fewer years of sulfa exposure, if we selectively under-estimate their fertility, we will tend to under-estimate the coefficient of interest. The estimates we show are, by this criterion, conservative.

the number of children ever born to a woman. The results are very similar using either measure, confirming a choice-led interpretation of our findings.

Results using net fertility are columns (4)-(6) in Table 2. The pattern of results is similar to that documented so far: pneumonia mortality decline is associated with a significant decline in fertility on both the extensive and intensive margins. In particular, an interquartile decline in pneumonia mortality (0.26), evaluated at the average number of reproductive years of exposure to sulfa drugs (14.9), led to 0.11 fewer children for the average woman, which is 5.7% of the pre-sulfa baseline mean, and a 1.4 percentage point increase in the probability of being childless, a 5.0% increase from baseline. Comparable estimates for gross fertility are in Table 3. These show that an interquartile decline in pneumonia mortality (0.26), evaluated at the average number of reproductive years of exposure to sulfa drugs, led to 0.081 fewer total births for the average woman, which is 3% of the baseline mean, and a 0.8 percentage point increase in the probability of being childless, which is 4.3% of the baseline mean.

Comparing the estimates for net fertility among women age 40-50 at enumeration with the estimates for fertility of women of childbearing age (18-40) from the preceding section (comparing columns (1)-(3) and (4)-(6) in Table 2), the coefficients for women who have completed fertility are consistently smaller than the coefficients for women who are of childbearing age. The estimates suggest that two thirds of the estimated impact of pneumonia mortality on childlessness in the childbearing-age sample is compensated delay, and that one third is a rise in permanent childlessness; the latter will incorporate both a decline in target fertility and uncompensated delay that may arise from biological decline in fecundity or from failure to find a marital match.

In the childbearing sample, we found a significant impact of maternal mortality decline on childlessness but in the sample of women with completed fertility, maternal mortality shows no significant impact on fertility. This suggests that the impacts of maternal mortality decline were only on fertility timing (advancement of fertility), while the pneumonia mortality decline influenced both the timing of fertility (consistent with the increased labor force participation of these women, which is discussed below) and target fertility (consistent with pneumonia mortality decline being concentrated among children, and thus increasing the incentive to invest in child quality).

We repeat the exercise for the distribution of fertility discussed above for completed net and gross fertility and find a similar pattern, particularly for the effect of reductions in pneumonia mortality on the distribution of completed net fertility (Figure A.4).

6.2.3 Nonparametric Patterns

We sought to look for the same patterns in a nonparametric specification, comparing average birth outcomes in states with above versus below median mortality in the pre-sulfa era for women aged 30-40 observed in census files. The broad patterns hold. See Figure A.5, which shows how, in the 1950 census and after, the more exposed above-median mortality states exhibit increases in childlessness, alongside lower intensive margin fertility, relative to below-median states.

6.3 Labor Market Outcomes

In this section we estimate the impacts of reductions in pneumonia and maternal mortality due to sulfa drugs on women's labor market choices. We cannot estimate dynamic models of the hazards of entry to and exit from the labor force or marriage because we do not have panel data on women; instead we have census data providing repeated cross-sections, and rely on sulfa exposure determined by birth cohort, as we did in the stock model of fertility.

As the census asks women about their current labor market status, we include all women aged 18-50 at the time of the census and 6-44 in 1937, pooling both childbearing women and women aged 40-50, for whom we estimated effects on completed fertility. Thus, the sample includes the birth cohorts 1893-1931. The results are very similar when widening the sample to include women aged up to 60, and narrowing the sample to women aged under 40; see Appendix F.

The results in Table 4 show that reductions in pneumonia mortality led to improved labor market outcomes for women by every indicator used, other than income. A decline in pneumonia mortality corresponding to an interquartile shift in the distribution, for a woman with the mean exposure to sulfa drugs in the estimating sample (18.3 years), is estimated to have increased the probability of being in the labor force of 2.6 percentage points and the probability of being employed of 2.8 percentage points. The average proportion of women who report being in the labor force (employed) in this sample is 37.1% (35.1%), so this increase is 7.0% (8.0%) of the baseline rate. This is of broadly similar magnitude to our estimate of the increase in the share of childless women and consistent with substitution between being in work and becoming a mother.[48] We explore this formally below by modelling the joint probability of being in the labor force and not having children.

The same decline in pneumonia mortality led to an increase in the occupation-based socioeconomic index of 6.6% relative to the baseline index score of 14.4. We obtain similar results when considering other occupational scores available in the census data, including *occscore* and the Duncan socioeconomic index. It increased weekly working hours by 1.15 hours, or 9% of the baseline mean, which implies an annual increase of 13.8 hours for the sample average of 12 working weeks per year.[49] We find no significant effect of pneumonia mortality reductions on personal income.

The fall in maternal mortality once again shows opposing effects to the fall in pneumonia mortality. A decline corresponding to an interquartile shift in the pre-sulfa distribution (-1.84) is estimated to have led to a decline in labor force participation of 3.8 percentage points (10.3% of baseline) and an increase in the probability of being employed of 2.6 percentage points (7.4% of baseline). It reduced working hours by 1.08 hours on average (8.4%), but had no effect on the

[48] If every additional childless woman worked (4.6pp from the net fertility measure), the increase in labor force participation would be 12.4% of baseline labor force participation. Thus, 7% underlines the plausibility of the link between changes in childlessness and employment of women.

[49] To put this in perspective, Bailey (2006) estimates an annual increase of 68 hours among cohorts with access to the birth control pill.

occupational score or income.[50,51]

An alternative explanation for our finding is that the labor market responses to exogenous changes in pneumonia mortality actually reflect responses to a woman's own risk of pneumonia, and not declines in child pneumonia mortality. This is unlikely given the low morbidity and mortality from pneumonia among prime-age adults, but possible if women overestimate the probability of death from the disease and/or the response is exquisite. To address this, we re-estimate our models replacing the core pneumonia measure with a measure of baseline pneumonia mortality rates among 25-35 year-old adults and a measure of baseline pneumonia mortality rates among children under five (Table A.14). We find that the coefficients on the adult rate are mostly insignificant and of the opposite sign, while the coefficients on the child rate are consistent with the main estimates. This indicates that improvements in child health were the driver behind the increase in labor force participation in response to pneumonia mortality decline that we estimate. Cross-sectional scatter plots for the US in 1930 also show an association of women's labor force participation with child mortality but not with adult mortality rates; see Section 7.

Together, these results show that reductions in pneumonia mortality increased the likelihood that women had careers. Relevant to understanding the big picture evolution of trends in women's labor force participation, we also find that reductions in maternal mortality reduced this likelihood.

Nonparametric Patterns We compare average female labor force participation rates in states with above versus below median rates of pneumonia mortality in Figure A.5: while the labor force participation rates converge until 1940, they diverge after that, with women in above-median mortality states participating in the labor force in greater numbers, consistent with the main findings.

The Joint Probability of Childlessness and Labor Force Participation Thus far we have demonstrated, using independent reduced form equations, that the sulfa-led reduction in pneumonia mortality led to lower total fertility, higher childlessness and higher rates of labor force participation, employment, working hours and occupational quality among women. We have argued, with reference to a stylized model, that the fertility and labor market responses are linked. In order to provide further evidence in support of this, we estimate the impact of sulfa-led mortality declines on the joint probability of being childless and being in the labor force (Table 5).

[50] Albanesi and Olivetti (2016) estimate that maternal mortality decline led to increased fertility, in line with our findings. However, in contrast to our results, they find that it led to an increase in female labor force participation. However, they develop a macro-model to study broad trends, so the studies are not strictly comparable. The authors present a theoretical model that focuses on the interaction of several direct drivers of the decline in the marginal cost of women's work, including maternal mortality decline and infant formula availability. Our theoretical framework assumes that child quality enters the utility function and that, as infant survival improves, women want fewer children, at least on the intensive margin.

[51] In general, whether maternal mortality decline leads to higher or lower labor force participation will depend on whether one is looking at cohorts exposed before or after they have completed their education. This is because maternal mortality decline will, by lengthening life expectancy, increase the returns to education. However, conditional on education, it will tend to increase fertility and thereby lower labor force participation.

Reductions in pneumonia mortality increased the probability that women were both working and childless by 2.7 percentage points (13% of the baseline probability of 20.5%); see column (1). It reduced the probability of not being in the labor force and having children by 3.2 percentage points, or 6.5% (column (4)), and did not change the probability of not working and being childless, or working and having children. We cannot reject that the coefficients in columns (1) and (4) are of the same magnitude but opposite sign, which indicates a direct substitution from stay-at-home mothers to working women without children.

It is also useful to explore the descriptive relationship between childlessness and labor force participation, tabulating labor market outcomes by childlessness status (Table A.3). Childless women were 34 percentage points more likely to be in the labor force, worked 12 additional hours per week, earned over $1000 more per year and had higher occupational scores than women who were mothers. We further report a descriptive regression of the relationship between labor force participation and childlessness, so as to condition upon birth cohort, state of birth and race, which produces similar results (Table A.15).

Education Choices In Appendix G we discuss estimates of the effect of sulfa-induced mortality decline on education choices. This is a natural complement to the labor market outcomes, though the fact that we consider women who were already of childbearing age in 1937 in our main analysis means that many of these women will have already completed their education. We find evidence that child (pneumonia) mortality decline led to an increase in high school completion rates in the subset of women who had not yet completed their education by 1937, but had no effect on the rate of college completion, while maternal mortality decline had the opposite effect. Our findings are consistent with Goldin, Katz, and Kuziemko (2006), who discuss the timing of the expansion of college education, and that this occurred later, among *children* of the sulfa cohorts.

6.4 Marriage Market Outcomes

We now consider the impact of sulfa exposure on marriage market outcomes. We use the specification in equation (7) and model three dimensions of marriage market outcomes: current marital status, whether the woman was ever married, and age at first marriage for ever married women. In the main estimates, we use the same sample as for labor market outcomes: 18 to 50 year old women at census enumeration and their ever married status. We report estimates for current marital status and age at first marriage for childbearing women (aged 18 to 40 at census) to capture any delay in marriage that may have mirrored fertility delay, and report estimates using alternative age-at-census samples in Appendix F.

The results are in Table 6. A reduction in pneumonia mortality of the size of an interquartile shift is estimated to have reduced the probability of women ever having married by 1.4 percentage points (1.65% of baseline mean), and to have reduced the probability of being married at the time of the census in the sample of women of childbearing age by 1 percentage point (1.5%). Thus, pneumonia decline led to both postponement of marriage and a reduction in marriage entry.

As for fertility and labor force participation, maternal mortality decline has the opposite effect to child mortality decline. We estimate that a reduction in maternal mortality corresponding to an interquartile shift increased the probability that a woman was married at the time of the census by 2.1 percentage points (2.9%), and the probability that she had ever married by 1.9 percentage points (2.3%). We find no significant impact of sulfa exposure on the average age at first marriage.

Baudin, de la Croix, and Gobbi (2019) propose a model of childlessness in which marriage is a key pathway. An important assumption in their model, which contrasts with ours, is that women have imperfect control over fertility. Child mortality acts to limit the fertility of women of low socioeconomic status. Thus, they argue, a reduction in child mortality will make less educated women less attractive on the marriage market, leading to increased childlessness and lower fertility. We assume perfect fertility control (see Section 2.2), and our model provides a lower bound on the impact of mortality decline on fertility.[52] In line with the predictions of Baudin, de la Croix, and Gobbi (2019), we find that child mortality decline is associated with lower chances of having ever married, alongside an increase in childlessness. However, the coefficients indicate small effects relative to the labor market responses, suggesting that marriage was one important margin of response, but that labor force participation, rather than marriage, was the main mediator of impacts on childlessness.

Nonparametric Patterns Figure A.5 compares the average marriage rates of women in above-median and below-median pneumonia mortality states: while marriage rates evolve in a parallel fashion up until 1940, the marriage rates of women in above-median mortality states fall after and remain lower than those of women in below-median mortality states. This is consistent with the estimates above.

6.5 Heterogeneity by Race and Education

We examine heterogeneity in outcomes by education and race and find that the compliers to pneumonia and maternal mortality decline were different groups of women. We summarize these results here; details are in Appendix E. First, consider impacts of pneumonia mortality decline. Fertility delay and the extensive and intensive margin responses were similar among women with higher education versus those who had not completed high school education. Labor market responses were also similar across the two education groups, though effects on marriage were concentrated among less educated women. That fertility and labor market responses were similar across the education distribution is pertinent as it undermines concerns about confounding from World War II-related labor market mobilization, which favored educated women (Goldin and Olivetti 2013). Also, the similar labor market responses at the two ends of the education distribution in response to child mortality decline suggests that our fertility delay hypothesis is a credible alternate explanation of the known U-shaped relationship between childlessness and education noted in the United States (Baudin, de la Croix, and Gobbi 2015).

[52] We thank David de la Croix for this insight.

With regards to race, fertility of white and black women responded similarly to falling pneumonia mortality rates, though response coefficients were larger for black women, likely owing to higher baseline rates of pneumonia. On the other hand, labor force participation and employment changed only among white women, while personal income increased only for black women. This pattern of results could be explained by higher baseline rates of labor force participation among black women (Boustan and Collins 2014), relegating any economic impacts to the intensive margin.

Maternal mortality decline had a significant effect only on the fertility choices of college-educated women. This lines up with a similar result in Albanesi and Olivetti (2014). We do not estimate any significant differences across education in the labor market impacts of maternal mortality decline. Maternal mortality decline impacts on fertility and the labor market were stronger for black women, consistent with their baseline mortality rates being higher.

6.6 Robustness checks

We have already documented robustness of the estimates to a range of control variables and specification checks. The pattern of results is similar in two different specifications and sample - the hazard and stock models, is similar irrespective of whether we use net or gross measures of fertility, and is not sensitive to changing the precise cohorts of women included in the sample. We have also shown that the broad patterns hold in non-parametric checks. This stability strengthens confidence in the results. In this Section, we nevertheless discuss additional specification checks. In Appendix F, we discuss robustness to further checks, including alternative sample definitions, adjustment of standard errors for multiple hypothesis testing, and exclusion of outlier states.[53]

Confounding Factors Inference with our research strategy relies on getting the timing of the introduction of antibiotics right. For this we rely on documentary evidence that the advent of sulfa drugs was widely publicized, for example in a *New York Times* article in December 1936 ("Conquering Streptococci"), and that historians have documented widespread uptake in 1937 (Lesch 2007). Figure A.6 shows extracts from two articles that appeared in the New York Times in 1936. The event study plots (Figure 7) discussed earlier also ratify the introduction of sulfa drugs in and not before 1937, as they show a fairly flat profile of impacts of pre-intervention mortality rates until 1937, after which there is a dip associated with pneumonia decline and a jump associated with maternal mortality decline.

Still, we may be concerned that these changes were driven by a coincident event rather than by the arrival of sulfa drugs. We therefore investigate robustness of our findings to accounting for events that occurred around the time of the sulfa intervention and that may have influenced fertility, labor market and marriage market outcomes. These are the New Deal, the Second World War, and the Dust Bowl, as well as the introduction of prescription charges for sulfa drugs in 1939. The New Deal was a government-funded program of spending and loans that aimed to tackle the

[53] For compactness, we report robustness estimates for completed fertility using gross fertility. The estimated impacts on completed net fertility are also robust to all listed checks.

effects of the Great Depression. We accessed data on state-year variation in New Deal spending (Fishback, Kantor, and Wallis 2003) and included this as a control, interacted with sulfa exposure ($post1937$ in the hazard model and $sulfayears$ in the stock model). The coefficients of interest are robust to this (Table 7 and Panel A, Table 8 for fertility outcomes, and Panel A, Tables 9 and 10, for labor and marriage market outcomes).

The United States entered the Second World War in December 1941. To assess the relevance of this, in the hazard model we restrict the sample to births occurring before 1942. In the stock model, we control for state-level troop deployment, obtained from Goldin and Olivetti (2013), interacted with individual exposure to the war, measured as the number of fertile years of the woman from 1942 onwards. This accounts for the possibility that war exposure affected lifetime fertility, and that state-cohort variation in war exposure is correlated with state-cohort variation in sulfa exposure. Our findings in both specifications are essentially unchanged (Table 7 and Panel B, Tables 8-10). Goldin and Olivetti (2013) show that WW2 affected the labor force participation of women at the upper end of the education distribution, while we find that the labor market impacts of the arrival of sulfa drugs were similar across education groups (Appendix E). This is additional evidence that the labor market impacts of sulfa drugs were not driven by WW2 mobilization.

The Dust Bowl refers to a period of drought and dust storms during the 1930s that damaged agriculture in several southern U.S. states and resulted in large out-migration from those states. To account for this, we estimate our models excluding states most affected by the Dust Bowl.[54] The results are, if anything, strengthened by the omission of the Dust Bowl states (Table 7 and Panel C, Tables 8-10). Sulfa drugs were available without prescription until 1939. Our findings are similar when we exclude all births after prescription was introduced (Table 7).

Omitted Trends The most likely candidate confounders in our setting are economic development and the disease epidemiology of the state. For instance, one may be concerned that if states with high pre-intervention disease burdens (the more treatable states in our set up) were also on a higher economic development trajectory, that it is this that generates our finding of higher labor force participation or lower fertility. Our main specification already controls for trends in 15 indicators of state-level income, infrastructure and disease prevalence, allowing these trends to break in 1937 and thereby subjecting our strategy to a stringent test (see 4). We now consider further checks on the role of omitted trends.

The main specification in the hazard model includes census region-year fixed effects. The estimates are similar when we include state linear trends or census division-year fixed effects instead (Table 7, columns (5) and (6)). We address the possible role of mean reversion by controlling for the state-level pre-sulfa average value of the outcome.[55] Table 7 and Panel D, Tables 8-10 show that the estimated coefficients are similar to those in the main results.

Next, we estimate impacts of a "placebo" intervention using an approach similar to that in

[54] These were Nebraska, Kansas, Colorado, New Mexico, Oklahoma and Texas.

[55] The outcomes are averaged over the 1900-1930 censuses and interacted with sulfa years in the stock model, and calculated for the years 1930-1936 and interacted with the post-1937 dummy in the hazard model.

Aaronson, Lange, and Mazumder (2014). This directly addresses concerns over omitted long-run trends in the stock model. We use data on individual outcomes in the 1910-1930 censuses, selected so that the outcomes were realized before the invention of sulfa drugs, and we create a fake intervention forty years previous to the true year (i.e. in 1897 rather than 1937), so that we have sufficient variation in placebo sulfa exposure in the census data. We then estimate the baseline specification of the stock model. The results in Panel E of Tables A.17-A.19 show that the coefficients on pneumonia and the maternal mortality rate are small and insignificant.

To verify the absence of diverging pre-trends in the hazard sample, we regress the probability of birth in the pre-sulfa era, 1930-1936, on a linear time trend interacted with a dummy variable equal to one for states with above median mortality, and zero otherwise. The results, in Table A.23, show no evidence of differential pre-trends across high and low mortality states in the hazard sample. In the hazard specification, Figure 7 showed a trend break in the probability of birth in 1937 and not elsewhere, which is similar to a "placebo" check.

In the hazard model, identification is relatively clean because the flow of births is analysed with reference to the discrete event of the introduction of sulfa drugs. However, in the stock model, individual years of exposure evolve linearly as a function of birth cohort, and are interacted with the pre-sulfa state-level disease burden for a sulfa-treatable disease. As a further check on this specification, we redefine the exposure variables to be binary, comparing the fully exposed with the unexposed, and omitting partially exposed women from the sample. The results are consistent with the main findings (Tables A.24-A.26).

Finally, to allow for the possibility that the underlying confounders are poorly measured (e.g. state income may not fully capture economic development), we follow Pei, Pischke, and Schwandt (2018) and perform a version of a balance test. We regress these potential confounders on the sulfa exposure variables.[56] Of the 22 coefficients (pneumonia and maternal mortality coefficients for 11 state- and time-varying control variables), only one is statistically significant, which is the coefficient on pneumonia exposure in the equation for tuberculosis mortality. However, investigating this by estimating an event study model for tuberculosis mortality decline, we see a secular decline with no break in 1937.

Measurement of Fertility and Survivorship Bias in Child Mortality There are two potential concerns with the way that we measure fertility: first, that we miss children who have left home; second, that the conception date is a more accurate measure of a woman's fertility choice than the birth date of the child. To address the first, we take two approaches. First, we follow Aaronson, Lange, and Mazumder (2014) and in our measures of net fertility only include children

[56] Specifically, we estimate variants of the regression

$$controlvar_{s,t} = \alpha + \beta_1 post1937 * prePnemonia_s + \beta_2 post1937 * preMMR_s + \beta_3 preNeumonia_s + \beta_4 preMMR_s + \boldsymbol{\beta}_5 \boldsymbol{\theta}_t,$$

where *controlvar* are all the 11 time-varying control variables we include on the right-hand side in our main estimates, and the time period is 1930-1943, as in the hazard model. There are fewer control variables than the total number of state controls, because for some we only have data for one year (literacy, FLFP, and the dates of birth and death registration entry).

aged below 10 (Panel F of Table A.17). The estimates are very similar to the main estimates. Second, in the hazard model, we only include potential births from the 1950 census that would have occurred in the years 1940-43: this way, the oldest child would have been ten. The estimated coefficients are robust to this sample restriction (Table A.16, column (1)). To address the second concern, we proxy a child's date of conception as one year before the date of birth, and re-estimate the hazard model using the date of conception as the outcome, rather than the date of birth (Table A.16, column (2)). Again, the estimates are unchanged.

When child mortality is high, the gross measure of fertility (children ever born to a woman) will overestimate surviving births. The net measure only counts children living with the mother, so it partially addresses this problem, but not entirely as children alive at the census date may subsequently die. The extent of overestimation will be correlated with pre-intervention pneumonia mortality, which is part of our exposure variable. Pneumonia mortality rates decline exponentially from birth to age five, after which they flatten out. Therefore, the fact that we find similar estimates when restricting our measure of fertility to children aged below 10 and when including all surviving children, shows that any survivorship bias is likely to play a limited role in our estimates.

Variation in Pneumonia and Maternal Mortality The maps in Figure A.2 show that pneumonia and maternal mortality were geographically dispersed, and did not vary in the same spatial pattern. However, the mortality rates are somewhat clustered in the south. Hence, we wish to ascertain that our results do not capture differences in trends between deep south states and the mountain west states. Table A.16, columns (3) and (4), show estimates of the hazard model where we omit the mountain west and deep south states in turn, while Tables A.17-A.19, Panels G and H, show the same exercise for the stock model estimates.[57] Dropping these groups of states does not substantially change the estimates.

In Appendix F, columns (2) and (3) in Table A.32 and Panels b and c in Tables A.33-A.35 show that the coefficients on pneumonia mortality decline are not significantly different when omitting maternal mortality decline.

Under-Reporting and Measurement Error in Pneumonia Mortality In Section 5, we explained that we use the all-age pneumonia and influenza mortality rate in place of the child pneumonia mortality rate to reduce measurement problems, arguing that the bulk of the change in the all-age pneumonia mortality rate after 1937 was driven by the steep decline in the infant rate (Figure 4). To assess the sensitivity of our estimates to this choice of data, we used the child (under-5) mortality rate from pneumonia and influenza instead. The results are in Table A.16 (column (5)) and Panel I, Tables A.20-A.22, and the main results are similar although the coefficients are reduced in magnitude, suggesting attenuation bias. Consistent with this, the coefficients increase in magnitude and precision when we estimate a 2SLS regression where the under-5 mortality rate is

[57] The mountain states are Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, and Wyoming. The deep south states are Louisiana, Mississippi, Alabama, Georgia, and South Carolina.

instrumented with the all-age rate used in the main estimates (Table A.16 (column (6)) and Panel J, Tables A.20-A.22).

Survivorship Bias - Maternal Mortality We face the common problem that we only observe survivors; mothers who died as a result of childbirth are not observed in census enumerations. It seems plausible to assume that women succumbing to maternal mortality tended to have higher fertility, and were in states with higher pre-intervention levels of maternal mortality. It follows that when these women are selected out of the fertility sample, we will tend to overestimate the increase in fertility that flows from the drop in maternal mortality.[58] This bias is mitigated by our controls for observable individual indicators of risk such as the age and education of the woman. For a sense of the extent of selection, see the mortality rate statistics in Table A.1.

Endogenous Migration If prospective mothers migrated in response to disease, then the introduction of sulfa drugs may have influenced migration patterns. In this case, we need to be sure that our findings do not reflect compositional change.

In our main estimation sample, we omit migrants (women whose birth state is not the same as their resident state). If we instead include these women, and if we also instrument for their mortality decline exposure in their state of residence using the mortality decline exposure of their birth state, the results are unchanged (Table A.16, columns (6)-(7), and Tables A.20-A.22, Panels K and L).

We also modelled migration as a function of post-sulfa declines in pneumonia and maternal mortality using two different indicators for migration. First, we defined an indicator for migrants as individuals for whom the birth state is different from the census enumeration state; second, we defined an indicator for migration between 1935 and 1940 using the information from the 1940 census. The estimates in Table A.27 show no evidence that sulfa-induced changes in mortality rates influenced migration.

7 Broader Relevance: Long-Run Patterns

We have demonstrated that in response to declines in pneumonia mortality, women had fewer children and were more likely to be childless. They also had higher labor force participation and lower marriage rates. We now demonstrate that this confluence of results is not an artefact of the sample, nor driven by a particular feature of the estimated specifications. We show that these correlations are evident in the raw data for the United States in 1930, as well as in contemporary data across African countries. Figure 9 plots the cross-sectional correlation across U.S. states in

[58] The positive correlation between fertility and maternal mortality implies that, pre-sulfa, high fertility women were more likely to die. Thus, observed fertility pre-sulfa for high mortality states is underestimated and the rise in fertility that we attribute to a reduction in maternal mortality may be partly due to high fertility women being more likely to survive post-sulfa.

1930 of pneumonia mortality with each of childlessness and total fertility. Childlessness is negatively correlated with pneumonia mortality, while total fertility is positively correlated with it.

We find a similar negative correlation between childlessness and other measures of child mortality, including under-2 diarrhea mortality and under-1 mortality from all causes - see Appendix H. In contrast, *adult* mortality rates, such as heart disease and cancer, are positively correlated with childlessness (and negatively correlated with total fertility). This is in line with our argument that the relationship between child mortality and childlessness is linked to the choice to delay fertility and continue in the labor market in the reproductive ages. Importantly, it does not support the competing hypothesis that childlessness results from improvements in women's health. Figure 9 also shows scatter plots of labor force participation and marital status against child mortality. Labor force participation is inversely correlated with child mortality (though less pronounced than childlessness), while marriage is positively correlated, consistent with our causal estimates.

Turning to contemporary data, Figure 10 plots the cross-country relationship between fertility, labor supply and marriage and infant mortality (from all causes), across African countries in 2015. Similar to the early 20th century data from the U.S., these contemporary data show a positive correlation of infant mortality with both total fertility and ever married rates, and a negative correlation of infant mortality with childlessness and labor force participation.

8 Conclusion

This paper presents quasi-experimental estimates of how improvements in child health and survival influenced fertility, with a particular focus on the timing of fertility, the behavior of the extensive margin response, and its relation to labor market choices and marriage among women. The estimates are relevant to debates concerning the trade-off between career and family, the rise of female labor force participation, and to models of the demographic transition and economic growth. The analysis produces three striking findings. First, a decline in child mortality (driven by pneumonia decline) led to fertility delay, a reduction in overall (and completed) fertility, with fewer women having three or more children, *and* an increase in childlessness. Second, the decline in pneumonia mortality was associated with a higher propensity to work, higher occupational scores, and a lower probability of having ever married. A third substantive finding is that the impact of maternal mortality decline on all of these outcomes, while less robust, was systematically in the opposite direction.

We argue that the first two findings are linked, namely, that child mortality decline made it rational to delay fertility, and that this led to the changes in labor market and marriage outcomes that we document. Reductions in child mortality reduced the time that women had to spend childbearing, and also reduced their target number of children by increasing child health and child quality. It also reduced the time that women, being main caregivers, spent caring for sick children. Together, this will have allowed women more time for productive activities. For women in the labor market, positive shocks to wages, negative shocks to fecundity or fertility preferences, or inertia,

can result in persistence of the childless state. We outline a dynamic model of fertility and labor market choices that shows a greater propensity for fertility delay and labor force participation in response to a decline in child mortality, when the joint probability of promotion at work and child mortality is low. The estimated patterns are consistent with this theory, fairly large, and robustly determined.

Our analysis has several attractive features: we model both the dynamics of fertility at the time of exposure to the reform and completed fertility, for the same cohorts of women observed in census data in different years. This allows us to estimate fertility delay, changed fertility targets and permanent childlessness. Also, we present estimates for both gross and net fertility, and allow pneumonia and maternal mortality to have independent impacts, and these are consistently in opposite directions for fertility, labor force participation and marriage. Thus, estimating impacts of overall mortality decline on fertility and labor market outcomes may overlook important causal relationships.

Most importantly, we provide new evidence on the drivers of childlessness and female labor force participation, and the need to consider fertility, labor market and marriage market choices in conjunction. No previous work appears to have proposed and tested the idea that child mortality decline may influence labor force participation and marriage decisions of women, by triggering fertility delay. These findings are relevant for contemporary development policy. Although there have been marked declines in child and maternal mortality in the last 25 years in response to worldwide mobilization and increasing investments in public health, there is limited causal evidence of fertility and labor market responses to these investments. Our findings suggest that investments in child mortality decline can contribute to the economic independence of women, where labor market opportunities are available such that women can delay childbearing and enter the labor market. Women's labor force participation and associated economic independence can lead to increased investments in children (Lundberg, Pollak, and Wales 1997, Baranov, Bhalotra, Biroli, and Maselko 2017) and a reduction in domestic violence (Aizer 2010).

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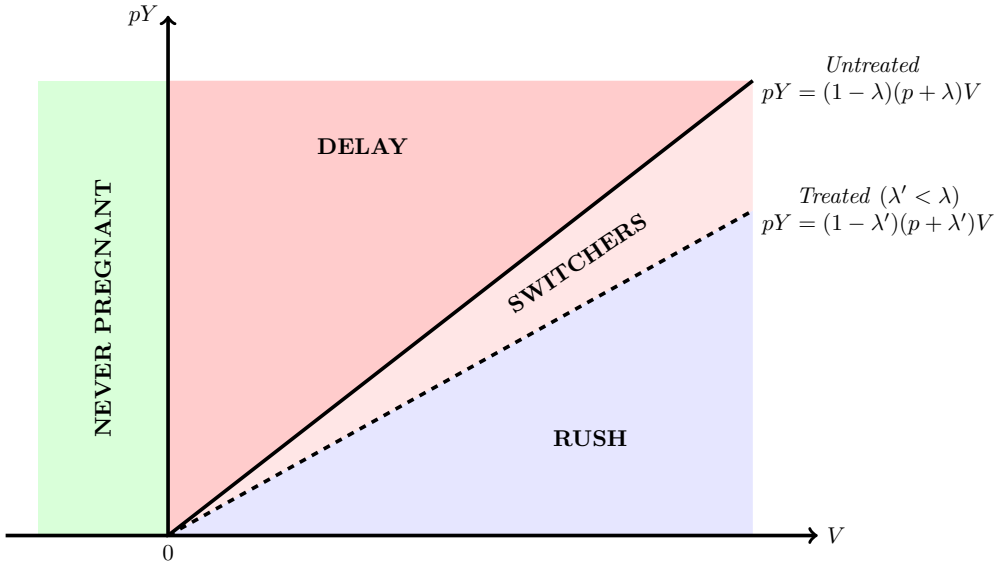
Tables and Figures

Figure 1: The effect of a reduction in child mortality λ on fertility delay in the model



The solid line delineates two groups of women: those above the line prefer to delay fertility and enter the labor market, while those below the line prefer to start childbearing in the first period. The line is given by the condition in equation (4). When child mortality falls, the solid line shifts down to the dotted line. More women prefer to delay instead of childbearing in the first period.

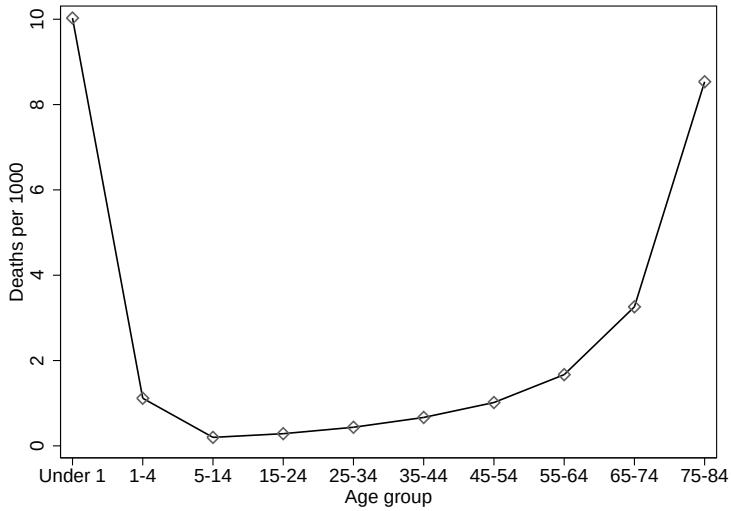
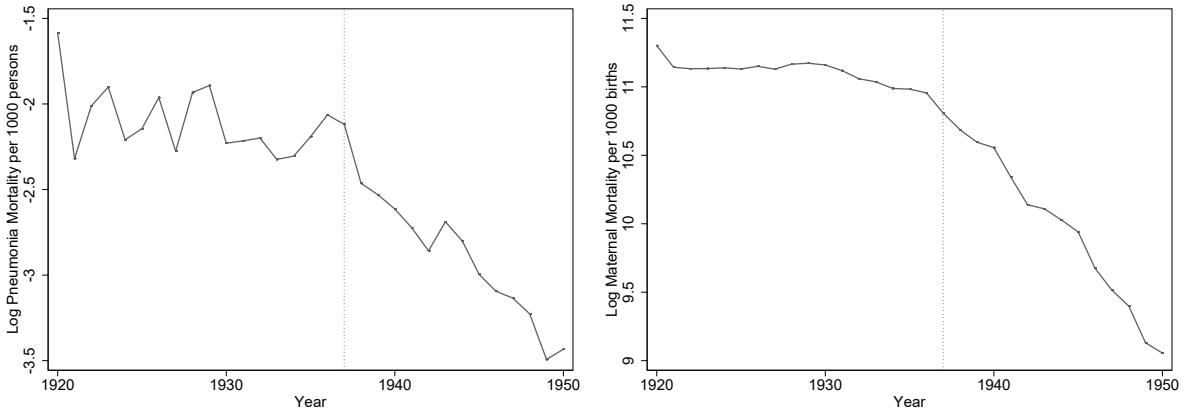


Figure 2: Pneumonia Incidence by Age, United States, 1935

This figure shows the average pneumonia mortality rate by age group in 1935 in the United States. Source: Britten (1942).



(a) Pneumonia Mortality (b) Maternal Mortality

Figure 3: Pneumonia Mortality and Maternal Mortality, United States

These figures show the average pneumonia mortality rate (left) and maternal mortality rate (right) in the United States over time. Source: Vital Statistics.

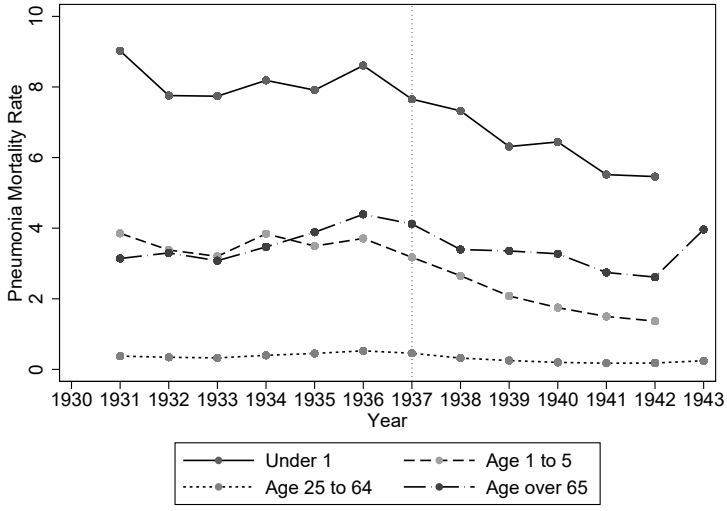
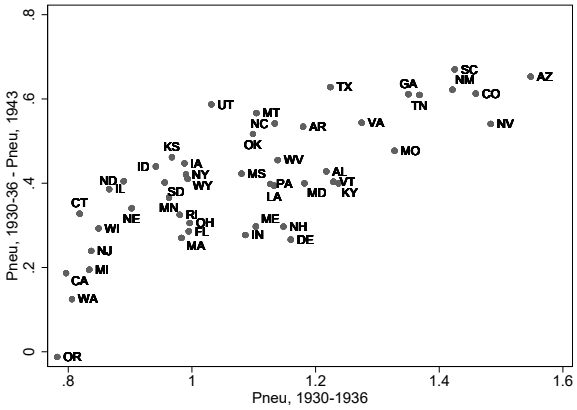
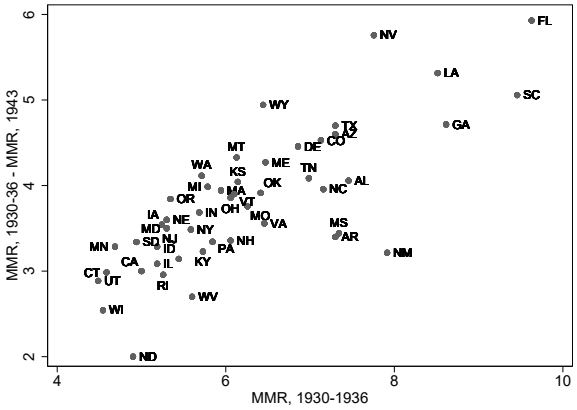


Figure 4: Pneumonia Mortality by Age, United States

This figure shows the average pneumonia mortality rate by age group and over time in the United States. Source: Vital Statistics.



(a) Pneumonia Mortality Convergence Post-1937



(b) Maternal Mortality Convergence Post-1937

Figure 5: Pneumonia Mortality and Maternal Mortality Convergence Post-1937, United States

These figures show the relationship between the 1937-1943 change and the 1930-1936 average level of pneumonia mortality (top) and maternal mortality (bottom) for different states in the United States. Source: Vital Statistics.

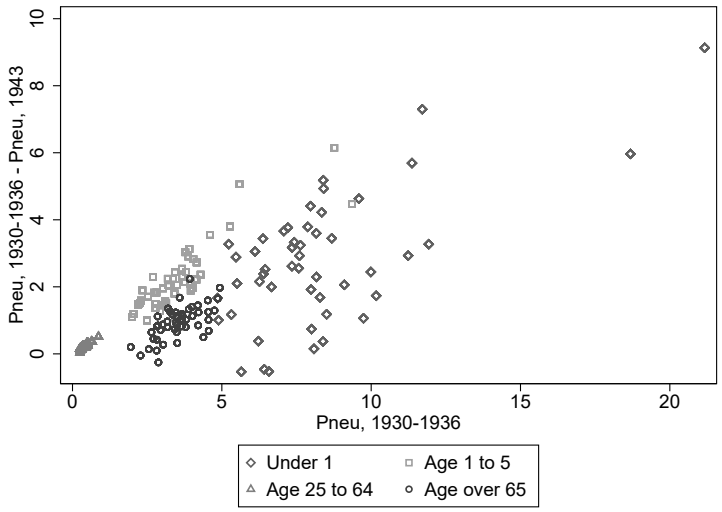
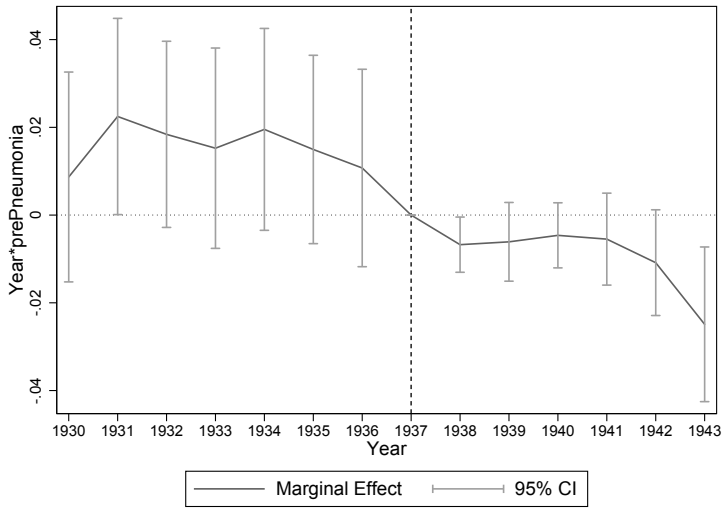


Figure 6: Pneumonia Mortality Convergence Post-1937 by Age, United States

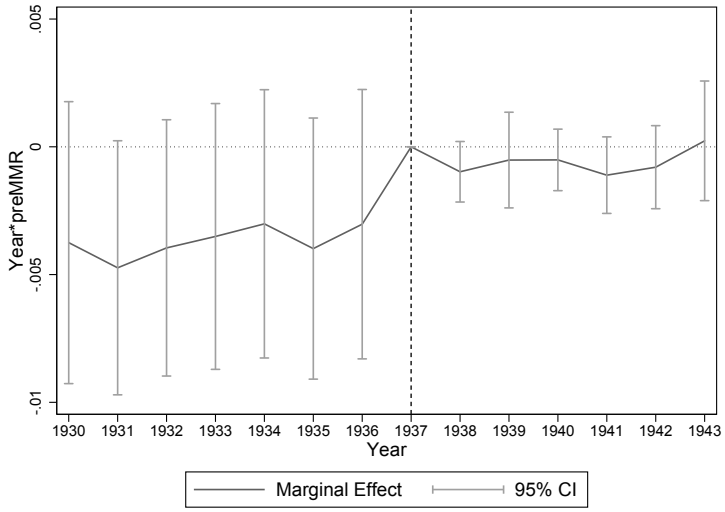
This figure shows the relationship between the 1937-1943 change and the 1930-1936 average level of pneumonia mortality in different age groups and different states in the United States. Source: Vital Statistics.

Figure 7: Event Studies

(a) Pneumonia Mortality



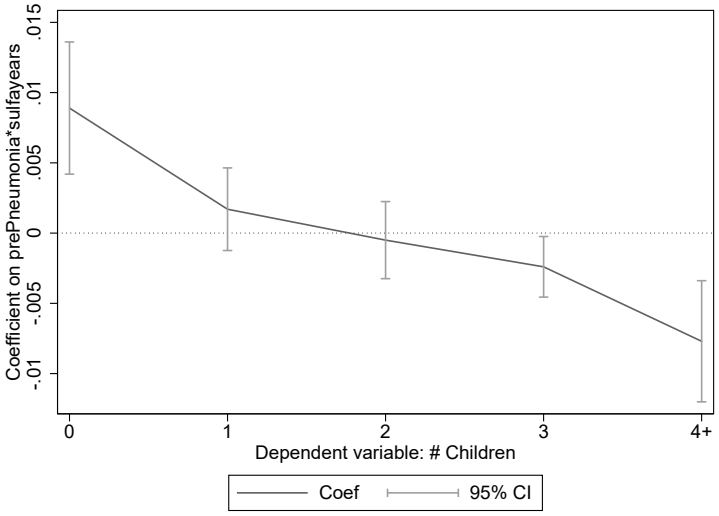
(b) Maternal Mortality



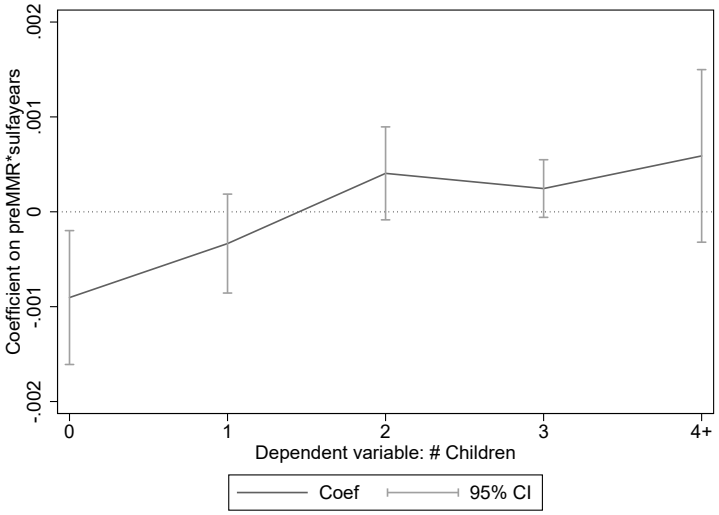
This figure displays the coefficients and 95% confidence intervals around these coefficients on the set of variables $prePneumonia * year$ (top) and $preMMR * year$ (bottom) where $year$ is a set of dummy variables for the 13 years 1930-1936 and 1938-1943 (1937 is the omitted case). The dependent variable is a dummy variable that equals one if the woman gave birth in that year, and zero otherwise. This is a Logistic regression. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, education, birth order and time since last birth fixed effects and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$.

Figure 8: Estimated Effects of Mortality Decline on the Fertility Distribution

(a) Pneumonia Mortality



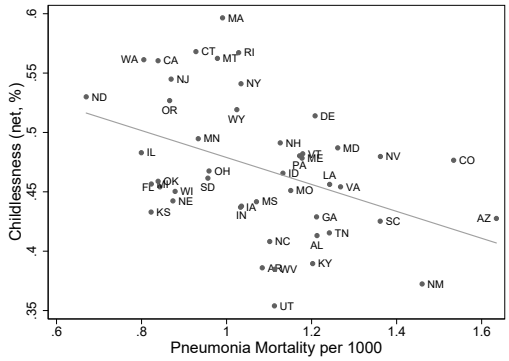
(b) Maternal Mortality



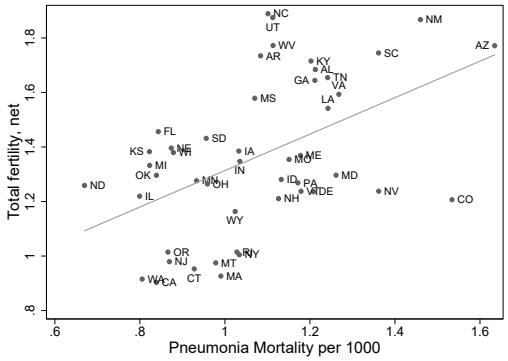
This figure displays the coefficients and 95% confidence intervals around these coefficients on the variable $prePneumonia * sulfayears$ (top) and $preMMR * sulfayears$ (bottom) in a set of five separate OLS regressions, where the dependent variables in these regressions are dummy variables for having no children in the household, exactly one child, exactly two children, exactly three children, and four or more children (from net fertility). Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for maternal mortality, malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$.

Figure 9: Pneumonia Mortality, Fertility, Labor Supply and Marriage in 1930 across US states

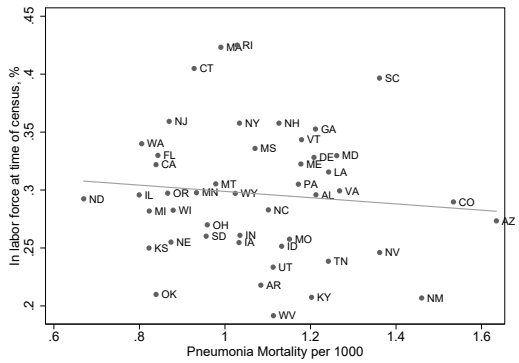
(a) Pneumonia Mortality and Childlessness



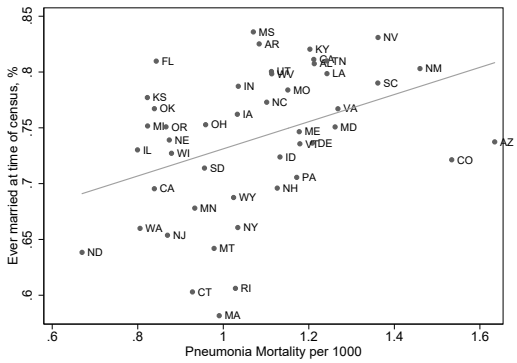
(b) Pneumonia Mortality and Total Fertility



(c) Pneumonia Mortality and Labor Force Participation



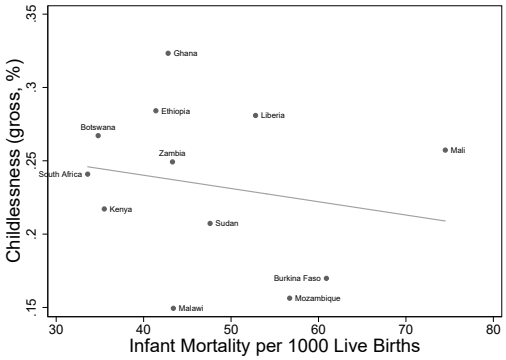
(d) Pneumonia Mortality and Ever Married Status



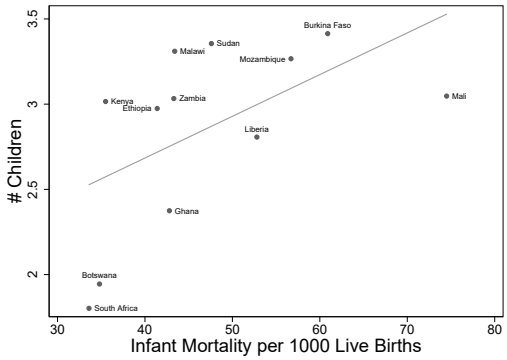
Figures are based on data from 18-50 year olds and fertility is net fertility, being based on information about children living in the household at the time of the survey. The upper age limit is chosen to minimise underreporting due to older children having left home. Still, to the extent that children have left home by the time a woman turns 50, we may underestimate fertility and overestimate childlessness, as is evident from the fairly high proportion of childless women in this age group. However, this is only an issue for the cross-sectional relationship if the age at which children leave home is correlated with underlying mortality rates. New Mexico appears to be an outlier in the pneumonia mortality data. In Appendix F, we show that the main results are robust to the exclusion of New Mexico from the sample.

Figure 10: Infant Mortality, Fertility, Labor Supply and Marriage, Africa, 2015

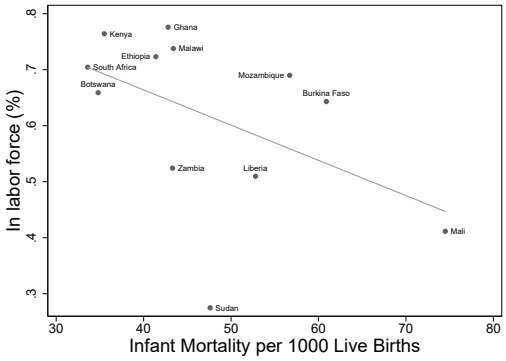
(a) Infant Mortality and Childlessness



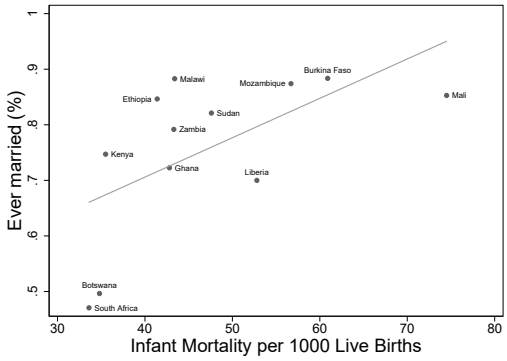
(b) Infant Mortality and Total Fertility



(c) Infant Mortality and Labor Force Participation



(d) Infant Mortality and Ever Married Status



The source of the fertility, labor market and marriage market data is the IPUMS International Database: all countries for which IPUMS data was available in 2000 or later are included, and all women aged 18-50 at the time of the census. We chose the census year closest to 2015 for each country. The mortality data are for 2015 and these data are sourced from UNESCO. Fertility is measured as gross fertility (births); childlessness is zero births.

Table 1: Probability of birth as a function of sulfa exposure, Logit

	(1) Birth	(2) Birth	(3) Extensive Margin	(4) Intensive Margin
$prePneumonia * post1937$	-0.0143***	-0.0233**	-0.0121**	-0.0095*
	(0.0033)	(0.0100)	(0.0060)	(0.0051)
$preMMR * post1937$	0.0009**	0.0031	0.0021	0.0007
	(0.0004)	(0.0024)	(0.0016)	(0.0010)
N	4558873	4499588	2894976	1604613
Mean	0.0865	0.0865	0.0513	0.1491
Controls				
Baseline controls	Y	Y	Y	Y
State characteristics	N	Y	Y	Y

The dependent variable is a dummy variable that equals one if the woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the woman's birth state level and the table shows marginal effects at the means of all covariates in the estimating sample. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1890-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, education, birth order and time since last birth fixed effects, year and census region*year fixed effects; column (2) adds state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s interacted with $post1937$, and income, public services, literacy, female labor force participation, and the year of state birth and death registration interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 2: Net fertility as a function of sulfa exposure

	(1)	(2)	(3)	(4)	(5)	(6)
	Childbearing women, age 18-40 at census			Completed fertility, age 40-50 at census		
	# Children	# Children | Children >0	Childless	# Children	# Children | Children >0	Childless
$prePneumonia * sulfayears$	-0.0483***	-0.0345***	0.0089***	-0.0212**	-0.0218**	0.0027*
	(0.0127)	(0.0117)	(0.0024)	(0.0103)	(0.0098)	(0.0016)
$preMMR * sulfayears$	0.0035	0.0009	-0.0009**	0.0006	-0.0001	-0.0001
	(0.0024)	(0.0026)	(0.0004)	(0.0021)	(0.0018)	(0.0003)
N	494437	313981	494437	237603	171166	237603
Mean	1.6590	2.6118	0.3648	1.9282	1.6760	0.2794

The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6), all based on net fertility (the number of own children living in the household). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census (columns 1-3) or 40-50 at the time of the census (columns 4-6), born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 3: Gross fertility as a function of sulfa exposure: Completed fertility

	(1) # Children	(2) # Children | Children >0	(3) Childless (0-1)
$prePneumonia * sulfayears$	-0.0209*	-0.0187*	0.0021**
	(0.0118)	(0.0106)	(0.0009)
$preMMR * sulfayears$	0.0007	0.0003	-0.0000
	(0.0023)	(0.0021)	(0.0001)
N	518933	421983	518933
Mean	2.5750	3.1660	0.1866

The dependent variable in column 1 is the total number of live births (gross fertility). The dependent variable in column 2 is the total number of live births conditional on having at least one (gross fertility). The dependent variable in column 3 is a dummy variable that equals one if a woman has no live births, and zero otherwise (gross childlessness). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and at least 40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 4: Labor market outcomes as a function of sulfa exposure

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1991**	7.3736	0.2421***
	(0.0017)	(0.0018)	(0.0771)	(15.3400)	(0.0652)
$preMMR * sulfayears$	-0.0008**	-0.0008**	0.0004	-0.1344	-0.0322***
	(0.0003)	(0.0003)	(0.0123)	(2.7242)	(0.0114)
N	727398	727398	517857	306280	727398
Mean	0.3510	0.3710	14.4093	1505.191	12.8097

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation, available for the 1950+ censuses; (4) the US Dollar amount of personal earnings in the past year, available for the 1950+ censuses; (5) hours worked in the past week, converted from intervalled data to a continuous measure using the midpoint of each interval. We find similar estimated effects of sulfa exposure on other measures of occupational score, including occscore (coefficients(standard error) 0.0775(0.0473) and -0.0146(0.0079) for $prePneumonia * sulfayears$ and $preMMR * sulfayears$ respectively), and the Duncan socioeconomic score (coefficients(standard error) 0.1512(0.0893) and -0.0139(0.0129) respectively). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 5: The joint probability of labor force participation and childlessness as a function of sulfa exposure

	(1)	(2)	(3)	(4)
	In Labor Force		Not in Labor Force	
	Childless	Not childless	Childless	Not childless
$prePneumonia * sulfayears$	0.0056***	-0.0001	0.0013	-0.0068***
	(0.0017)	(0.0010)	(0.0012)	(0.0019)
$preMMR * sulfayears$	-0.0007***	-0.0001	0.0002	0.0006***
	(0.0003)	(0.0002)	(0.0002)	(0.0003)
N	727398	727398	727398	727398
Mean	0.205	0.166	0.140	0.489

The dependent variable is the joint status of labor force participation and childlessness (based on net fertility) at the time of census enumeration. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility, labor and marriage market outcomes of women aged 18 to 50 at the time of the census, aged 6 to 44 in 1937, and born in the United States. The cohorts in this table were born in the years 1893 to 1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s interacted with $sulfayears$, income and public services, literacy, female labor force participation, and the year of state birth and death registration interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 6: Marriage market outcomes as a function of sulfa exposure

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0023*	-0.0032**	0.0021
	(0.0012)	(0.0012)	(0.0243)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0053
	(0.0002)	(0.0002)	(0.0055)
N	494437	727398	116632
Mean	0.7258	0.8499	21.1798

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage for woman who have ever married. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 6-44 in 1937 and 18-40 for columns 1 and 3 and 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 for columns 1 and 3 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 7: Probability of birth as a function of sulfa exposure - robustness checks

	(1) WW2	(2) New Deal	(3) Dust Bowl	(4) Excl post-1939 Birth	(5) Lin trend	(6) Division*year	(7) Mean Rev
$prePneumonia * post1937$	-0.0239**	-0.0247**	-0.0247**	-0.0256**	-0.0218*	-0.0288***	-0.0251**
	(0.0105)	(0.0107)	(0.0106)	(0.0113)	(0.0127)	(0.0092)	(0.0103)
$preMMR * post1937$	0.0041	0.0031	0.0048*	0.0048***	0.0031	0.0036**	0.0029
	(0.0026)	(0.0024)	(0.0026)	(0.0028)	(0.0031)	(0.0017)	(0.0023)
N	4053834	4499588	4053176	3417407	4499588	4499588	4499588

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. The robustness check in each column is described in detail in Section 6.6. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1890-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects (except for columns (5) and (6), which replace these with state linear trends and census division-year fixed effects respectively), as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 8: Fertility outcomes as a function of sulfa exposure - robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel A (New Deal)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0417***	-0.0316**	0.0074***	-0.0210	-0.0186	0.0020*
	(0.0140)	(0.0125)	(0.0024)	(0.0134)	(0.0119)	(0.0010)
$preMMR * sulfayears$	0.0033	0.0008	-0.0009**	0.0007	0.0003	-0.0000
	(0.0024)	(0.0026)	(0.0003)	(0.0023)	(0.0021)	(0.0002)
N	494437	313981	494437	518933	421983	518933
Panel B (WW2)						
$prePneumonia * sulfayears$	-0.0442*	-0.0218	0.0113***	-0.0172	-0.0150	0.0020**
	(0.0235)	(0.0189)	(0.0039)	(0.0115)	(0.0103)	(0.0009)
$preMMR * sulfayears$	0.0003	-0.0033	-0.0013**	0.0005	0.0001	0.0000
	(0.0038)	(0.0035)	(0.0006)	(0.0021)	(0.0019)	(0.0001)
N	317789	230684	317789	518832	421898	518832
Panel C (Dust Bowl)						
$prePneumonia * sulfayears$	-0.0499***	-0.0358***	0.0092***	-0.0267**	-0.0233**	0.0025**
	(0.0127)	(0.0123)	(0.0022)	(0.0117)	(0.0105)	(0.0010)
$preMMR * sulfayears$	0.0033	0.0001	-0.0010**	0.0011	0.0006	-0.0000
	(0.0023)	(0.0024)	(0.0004)	(0.0022)	(0.0020)	(0.0001)
N	444734	280263	444734	463435	376022	463435
Panel D (Mean reversion)						
$prePneumonia * sulfayears$	-0.0570***	-0.0416***	0.0106***	-0.0234*	-0.0197	0.0021**
	(0.0123)	(0.0124)	(0.0020)	(0.0130)	(0.0118)	(0.0009)
$preMMR * sulfayears$	0.0050**	0.0019	-0.0013***	0.0010	0.0004	-0.0000
	(0.0024)	(0.0026)	(0.0004)	(0.0024)	(0.0022)	(0.0002)
N	494437	313981	494437	518933	421983	518933

See notes to Table 2 for definitions of outcomes. The robustness checks are described in Section 6.6. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the U.S. and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940-1970 US decennial population censuses. See notes to Table A.18 for further details on estimation and control variables. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 9: Labor outcomes as a function of sulfa exposure - robustness checks

Panel A (New Deal)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0055***	0.0051***	0.3308**	29.8135***	0.2295***
	(0.0016)	(0.0017)	(0.1472)	(10.2308)	(0.0637)
$preMMR * sulfayears$	-0.0008**	-0.0008**	0.0218	-0.5819	-0.0320**
	(0.0003)	(0.0003)	(0.0292)	(1.6195)	(0.0114)
N	727398	727398	247015	306280	727398
Panel B (WW2)					
$prePneumonia * sulfayears$	0.0070***	0.0066***	0.3695***	14.5503	0.2931***
	(0.0017)	(0.0017)	(0.1293)	(14.5801)	(0.0533)
$preMMR * sulfayears$	-0.0003	-0.0002	0.0176	-0.6242	-0.0113
	(0.0003)	(0.0003)	(0.0271)	(2.6723)	(0.0092)
N	517746	517746	246952	306209	517746
Panel C (Dust Bowl)					
$prePneumonia * sulfayears$	0.0057***	0.0053***	0.3128*	1.7650	0.2395***
	(0.0017)	(0.0018)	(0.1627)	(14.9465)	(0.0639)
$preMMR * sulfayears$	-0.0009***	-0.0009***	0.0218	0.4506	-0.0375**
	(0.0003)	(0.0003)	(0.0313)	(2.3797)	(0.0113)
N	654187	654187	223428	274954	654187
Panel D (Mean reversion)					
$prePneumonia * sulfayears$	0.0059***	0.0053***			
	(0.0017)	(0.0017)			
$preMMR * sulfayears$	-0.0007**	-0.0007**			
	(0.0003)	(0.0003)			
N	727398	727398			

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, converted from intervalled data to a continuous variable using the midpoints of each interval. The robustness checks in the different panels are described in Section 6.6. See notes to Table A.18 for further details on sampling, control variables and estimation. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 10: Marriage market outcomes as a function of sulfa exposure - robustness checks

Panel A (New Deal)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0020	-0.0024*	0.0152
	(0.0013)	(0.0012)	(0.0245)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0050
	(0.0002)	(0.0002)	(0.0050)
N	494437	727398	116632
Panel B (WW2)			
$prePneumonia * sulfayears$	-0.0051**	-0.0042***	-0.2328
	(0.0025)	(0.0014)	(0.2526)
$preMMR * sulfayears$	0.0013***	0.0005**	-0.0188
	(0.0004)	(0.0002)	(0.0372)
N	317789	517746	81854
Panel C (Dust Bowl)			
$prePneumonia * sulfayears$	-0.0023*	-0.0031**	-0.0071
	(0.0012)	(0.0012)	(0.0260)
$preMMR * sulfayears$	0.0005**	0.0005***	0.0037
	(0.0002)	(0.0002)	(0.0065)
N	444734	654187	103929
Panel D (Mean reversion)			
$prePneumonia * sulfayears$	-0.0023*	-0.0031**	0.0157
	(0.0012)	(0.0012)	(0.0233)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0039
	(0.0002)	(0.0002)	(0.0046)
N	494437	727398	116632

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage for ever married women. The robustness checks in the different panels are described in Section 6.6. See notes to Table A.19 for details on sampling, control variables and estimation. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Appendix for Online Publication
Fertility and Labor Market Responses to Reductions in Mortality

Sonia Bhalotra
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A Data Description

The **mortality data** is extracted from US Vital Statistics (Grove and Hetzel 1968, Linder and Grove 1947, Ruggles, Alexander, Genadek, Goeken, Schroeder, and Sobek 2010, Bureau 1943). In particular, we combined and extended the data series collected by Grant Miller (http://www.nber.org

/data/vital-statistics-deaths-historical/), and by Seema Jayachandran, Adriana Lleras-Muney, and Kimberly Smith (http://www.aeaweb.org/articles.php?doi=10.1257/app.2.2.118).

State time series data on logged state per capita income were downloaded from the Bureau of Economic Analysis website (http://www.bea.gov/regional/spi/). Data on the number of schools, doctors, hospitals, and educational expenditures per capita were taken from Adriana Lleras-Muney's website (http://www.econ.ucla.edu/alleras/research/data.html). These data were originally collected from various volumes of the Biennial Survey of Education (schools and expenditures) and the American Medical Association's American Medical Directory (doctors and hospitals). For state per capita health expenditures, we used data collected from various reports from the US Census Bureau. (See http://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/6304?archive=ICPSR&q=6304). The state level data is matched to individual data by women's birth state.

The **main outcome variables** are constructed as follows.

- *Net total fertility* is the total number of own children living in the household. *Net childlessness* is a variable equal to one when this is zero and equal to zero otherwise.
- *Gross total fertility* is the total number of live births the woman ever had. *Gross childlessness* is a variable equal to one when this is zero and equal to zero otherwise. The number of live births was a question asked to ever-married women in the 1940 and 1950 censuses and to all women in subsequent censuses.
- The *intensive* margin of fertility for both of these measures is defined as total fertility conditional on not being childless; hence, this variable takes a missing value for childless women.
- The variable *Working* takes a value of one if the woman reports working at the time of the census and zero otherwise.
- The variable *In Labor Force* takes a value of one if the woman reports she is in the labor force at the time of the census.
- *Personal income* is the reported own income from all sources in the last year. It is available for the 1950 census and onwards.
- The *Hauser and Warren Socioeconomic Index (H-W SEI)* is a measure of occupational status based on earnings and education. It assigns a measure of prestige to each occupation. See ipums.org for a detailed explanation of its construction. It is available for the 1950 census and onwards. We also considered *occscore* from the IPUMS data and the *Duncan socioeconomic score* as outcomes, with similar results.
- *Hours worked* is the reported number of hours worked in the past week. The original data is an intervalled variable and it is converted to a continuous variable using the midpoint of each interval.

- The variable *Currently married* takes the value one if a woman is married at the time of the census and zero otherwise.
- *Ever married* is a dummy variable equal to one if a woman has been married at some point in her life and zero otherwise.
- *Age at 1st marriage* is the age at which a woman first married, only defined for women who have ever married, and not available for the 1950 census, hence making the sample size for this variable smaller than for the other outcomes.

Table A.1: Descriptive statistics: Control variables and Hazard data

Variable	Mean	Standard deviation
Control variables		
$prePneumonia$	1.0918	0.1989
$preMMR$	6.2610	1.2403
$preDiarrhea$	8.1358	5.7157
$preMalaria$	34.1667	70.4349
$preCancer$	0.9674	0.3109
$preHeartDisease$	2.1483	0.6439
$preTuberculosis$	0.6284	0.3616
$ln(Income\_per\_capita)$	5.9551	0.3960
$ln(Number\_of\_schools\_per\_capita)$	0.7586	0.6491
$ln(Number\_of\_hospitals\_per\_capita)$	-2.80	0.4427
$ln(Number\_of\_doctors\_per\_capita)$	0.1246	0.2291
$ln(Education\_expend\_per\_capita)$	4.6150	0.3887
$ln(Health\_expend\_per\_capita)$	-1.2317	0.6275
$Year\_of\_birth\_registration$	1921.17	5.3726
$Year\_of\_death\_registration$	1910.681	13.478
$Literacy$	0.9760	0.0374
$Female\_LFP$	0.1971	0.0596
N	48	
Hazard data		
Birth	0.0865	0.2811
$post1937$	0.5001	0.5
$Current\_birth\_order$	1.7182	1.2364
$Years\_since\_last\_birth$	6.8448	6.1723
$Birth\_year\_of\_woman$	1910.724	98.0187
N	4559108	

This table shows the mean and standard deviation of state level characteristics that are interacted with $post1937$ in the hazard sample and $sulfayears$ in the stock sample and included as control variables (top panel), and of outcome and control variables in the hazard model (bottom panel). The mortality rates from diseases are the average between 1930-1936, per 1000 population (or 1000 live births in the case of MMR), and all other variables are measured in 1930, except the year of entering the birth and death registration systems.

Table A.2: Descriptive statistics: Stock model

Variable	Mean	Standard deviation	N
Net Fertility (childbearing sample)			
# Children	1.6590	1.8316	496783
# Children | Children>0	2.6118	1.6712	315548
Childless (0-1)	0.3648	0.4814	496783
$Sulfayears$	20.0	6.0626	496783
Net Fertility (completed fertility sample)			
# Children	1.9282	1.9473	239432
# Children | Children>0	2.6760	1.8060	172524
Childless (0-1)	0.2794	0.4487	496783
$Sulfayears$	14.6401	8.8397	239432
Gross Fertility (completed fertility sample)			
# Children	2.5750	2.2927	520591
# Children | Children>0	3.1660	2.1428	423423
Childless (0-1)	0.1866	0.3896	520591
$Sulfayears$	14.8679	8.6624	520591
Labor Market			
Working (0-1)	0.3510	0.4773	730498
In labor force (0-1)	0.3710	0.4831	730498
Hauser-Warren SEI	14.4093	17.181	519972
Personal income	1505.191	2817.12	307378
Hours worked	12.8097	19.1029	730498
$Sulfayears$	18.2949	7.5187	730498
Marriage Market			
Currently married	0.7258	0.4461	496783
Ever married	0.8499	0.3572	926552
Age at 1st marriage	21.1798	3.4153	106814
$Sulfayears$	17.5947	7.9902	926552
Age at birth			
Age at 1st birth	24.0750	4.9714	440156
Age at 2nd birth	26.7165	5.0326	316185
Age at 3rd birth	28.6299	5.0395	183840
Age at 4th birth	30.1623	4.9682	101896

This table shows the mean and standard deviation of key variables in the stock model. Our dataset is a cross-section of fertility, labor and marriage outcomes of women aged 5-44 in 1937 and 18-40 (net childbearing fertility, current marital status), at least 40 (net completed, gross fertility), 18-50 (labor, age at birth, ever married) at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 (1900-1931 for net fertility and current marriage) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses.

Table A.3: Descriptive outcomes by childlessness status

Outcome	Childless women			Not childless women		
	Mean	St.dev.	N	Mean	St.dev.	N
Working	0.56	0.50	251652	0.24	0.43	478846
In labor force	0.59	0.49	251652	0.25	0.44	478846
H-W SEI	21.10	17.46	147691	11.76	16.33	372281
Personal income	2295.83	3328.75	88539	1185.31	2511.73	218839
Hours worked	21.06	21.05	251652	8.37	16.33	478846
Currently married	0.43	0.50	251652	0.92	0.27	478846
Ever married	0.51	0.50	251652	0.99	0.07	478846
Age at 1st marriage	21.01	5.38	50079	20.92	3.92	150534
Graduated from HS	0.25	0.44	251652	0.21	0.41	478846
Attended some college	0.12	0.32	251652	0.08	0.27	478846

This table shows the mean and standard deviation of outcome variables by (net) childlessness status in the stock model. All differences in means between childless and not childless women are statistically significant at the 1% level. Our dataset is a cross-section of fertility, labor market and marriage market outcomes for women aged 6 to 44 in 1937 and 18 to 50 at the time of the census, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses.

B Trends in Labor Force Participation, Fertility and Marriage

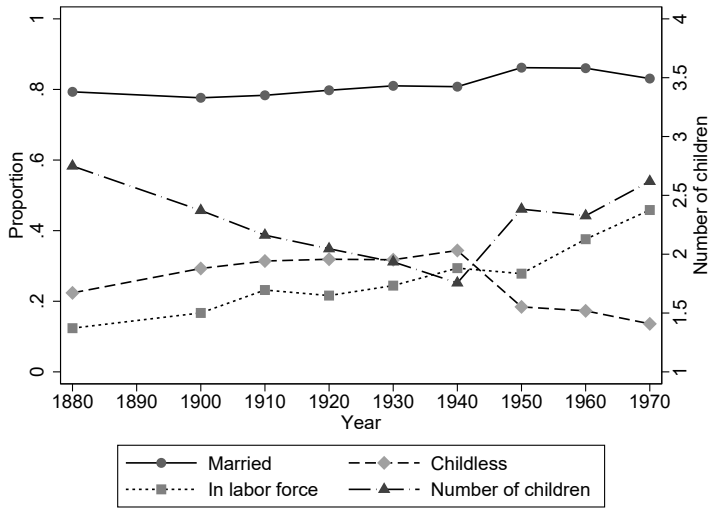


Figure A.1: Trends in childlessness, fertility, labor force participation and marriage, United States, 1880-1970

This figure is constructed from the US decennial population censuses 1880-1970. The sample consists of all women aged 30 to 40 at the time of the census interview and born in the US. Childlessness and total fertility is defined based on net fertility (children living in the home), and marriage and labor force status refer to these statuses at the time of the census.

C Trend Breaks and Cross-State Convergence

These tables formally test convergence in mortality rates after the introuduction of sulfa drugs in 1937. Table A.4 tests for the existence of a trend break in mortality rates in 1937, captured by a linear trend interacted with a post-1937 dummy variable. Table A.5 shows that high mortality states pre-1937 had larger declines in mortality rates post-1937.

Table A.4: Trend breaks in mortality rates

	(1)	(2)	(3)	(4)
	Levels		Logs	
	$\Delta Pneumonia$	ΔMMR	$\Delta Pneumonia$	ΔMMR
$year * post1937$	-0.0999***	-0.2143***	-0.1110***	-0.0930***
	(0.0059)	(0.0252)	(0.0059)	(0.0058)
$post1937$	-0.1408***	-0.5128***	-0.1287***	-0.0930***
	(0.0240)	(0.1021)	(0.0238)	(0.0235)
$year$	0.0192***	-0.2154***	0.0153***	-0.0335***
	(0.0042)	(0.0180)	(0.0042)	(0.0041)
N	667	667	667	667
R^2	0.7573	0.8981	0.7988	0.8944

These are OLS regressions (standard errors in parentheses) at the state-year level. The dependent variables are the year-on-year change in *Pneumonia*, the state-year average mortality rate from pneumonia, and the y-o-y change in MMR, the state-year average maternal mortality rate. The regressions also include state fixed effects. *year* is a linear time trend and *post*1937 is a dummy variable for the years 1937 and later. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.5: Test of convergence in state mortality rates

	(1) *Pneumonia*	(2) *MMR*
$prePneumonia * post1937$	-0.2940***	
	(0.0459)	
$preMMR * post1937$		-0.2234***
		(0.0396)
N	667	667
R^2	0.8603	0.9067

These are OLS regressions (standard errors in parentheses) at the state-year level. The dependent variables are *Pneumonia*, the state-year average mortality rate from pneumonia, and MMR, the state-year average maternal mortality rate. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. The regressions also include state and year fixed effects. *denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

D Theoretical Framework

D.1 Classical Quantity-Quality Model of Fertility

Consider the canonical quality-quantity model of fertility, following Becker and Lewis (1973) and Galor (2012). A woman derives utility $U(c, n, e)$ from consuming c and having n children with quality e. To simplify the exposition, we assume that utility is quasilinear[59], with

$$U(c, n, e) = u(n, e) + c,$$

where $u(0, e) = 0$. Her budget constraint is

$$n(\tau^q + \tau^e e) + c \leq I, \tag{8}$$

where I denotes her lifetime income, τ^q is the price of quantity, and τ^e is the per-child price of quality. The maximized value of having at least one child is

$$\max_{c,e,n\geq 1} U(c, n, e) \text{ subject to (8)},$$

[59] The effect of assuming quasilinear utility is to remove an income effect where a rise in income increases the demand for both quality and quantity, generating a positive relationship between income and the number of children. Becker and Lewis (1973) argue that the income elasticity of quality is higher than the income elasticity of quantity, such that these income effects do not arise in practise. In our setting, an advantage of using this specification is that it allows us to shut down a mechanism that is key to the process described in Baudin, de la Croix, and Gobbi (2015) and Baudin, de la Croix, and Gobbi (2019), where childlessness is associated with poverty through low fecundity and low marriage prospects. We thank Uwe Sunde for this insight.

which is a decreasing function of prices $\tau = (\tau^q, \tau^e)$. On the intensive margin of fertility, quantity and quality tend to be substitute goods, assuming that the income effect is not large enough to dominate the substitution effect. Thus, when the price of child quality declines, parents will substitute out of quantity and into quality (have fewer children). The key insight of Aaronson, Lange, and Mazumder (2014) is that the extensive margin response to a decline in the price of child quality and quantity will always be positive. This is because the value of being childless is simply $U(I, 0, 0)$ and therefore independent of prices; there is no substitution at the corner solution. The introduction of sulfa drugs reduced child mortality and morbidity, lowering the price of child quality and quantity. Intensive margin fertility can therefore have decreased or increased, depending on which price reduction dominated. On the extensive margin, however, the prediction is unambiguous that childlessness should have declined.

D.2 Proof of Proposition 1

Proof. The proportion of women who have one child is

$$\begin{aligned} C(\lambda, \tau) &= \delta(\lambda, \tau)(1-p)(1-\lambda) + (1 - \delta(\lambda, \tau) - \eta(\tau))(1-\lambda^2) \\ &= 1 - \lambda^2 - \delta(\lambda, \tau)(1-\lambda)(p+\lambda) - \eta(\tau)(1-\lambda^2). \end{aligned}$$

The total population effect of a decline in child mortality on fertility is $-\frac{dC(\lambda,\tau)}{d\lambda}$. This can be decomposed into the direct effect of λ and the indirect effect through prices τ:

$$-\frac{dC(\lambda,\tau)}{d\lambda} = -\frac{\partial C(\lambda,\tau)}{\partial \lambda} + \frac{\partial C(\lambda,\tau)}{\partial \tau} \cdot \left(-\frac{d\tau}{d\lambda}\right).$$

The price effect (the second term) is always positive. To see this, note that

$$\frac{\partial C(\lambda,\tau)}{\partial \tau} = \left(-\frac{\partial \delta(\lambda,\tau)}{\partial \tau}\right)(1-\lambda)(p+\lambda) + \left(-\frac{\partial \eta(\tau)}{\partial \tau}\right)(1-\lambda^2).$$

Since a fall in prices increases the value of getting pregnant, and decreases the net value of delay, it is easy to see that $-\frac{\partial \delta(\lambda,\tau)}{\partial \tau} < 0$ and $-\frac{\partial \eta(\tau)}{\partial \tau} < 0$. It follows that $\frac{\partial C(\lambda,\tau)}{\partial \tau} < 0$. Then, as long as the prices of quality and quantity are non-increasing in response to decreased mortality, we have $-\frac{d\tau}{d\lambda} \leq 0$ and

$$\frac{\partial C(\lambda,\tau)}{\partial \tau} \cdot \left(-\frac{d\tau}{d\lambda}\right) > 0.$$

Thus, the price effect leads to higher fertility and fewer childless women. This formalizes our intuition that, in line with Aaronson, Lange, and Mazumder (2014), price effects in response to sulfa drugs are unlikely to explain the increased childlessness that we find in the data.

The direct effect of decreased mortality on fertility can be further decomposed as

$$-\frac{\partial C(\lambda,\tau)}{\partial \lambda} = \underbrace{2\lambda(1-\delta(\lambda,\tau)-\eta(\tau))+(1-p)\delta(\lambda,\tau)}_{\text{mechanical effect}} - \underbrace{(1-\lambda)(p+\lambda)\left[-\frac{d\delta(\lambda,\tau)}{d\lambda}\right]}_{\text{dynamic effect}}.$$

The mechanical effect is positive. More pregnancies are successful, so there are more children. However, the dynamic effect can be negative, and offset the mechanical and price effects, if more women delay in response to the change, that is if $-\frac{d\delta(\lambda,\tau)}{d\lambda} > 0$. It is easy to see that this effect has the same sign as the effect on the marginal woman's incentives:

$$-\frac{d\delta(\lambda,\tau)}{d\lambda} \overset{\text{sign}}{=} -\frac{dN(\lambda,\tau)^i}{d\lambda}\bigg|_{N^i=0} = V^i\left(1-2\lambda-p\right).$$

Then the dynamic effect is negative if and only if

$$\lambda < \frac{1-p}{2}.$$

■

D.3 Modelling Other Shocks

In the main text, we analyzed impacts of a positive labor market shock. A similar analysis applies if we consider other sources of new information that can lead to persistence of the childless state, conditional on fertility delay. We sketch the intuition here. First, consider learning about the future benefits of work (Fernández 2013). In our framework, a simple way to model this is to suppose that with a certain probability, the woman learns at the end of the first period that her utility from work in the second period will be higher than her current utility (if this does not happen, then her utility from work in the second period stays the same). This is similar to the effect of job promotion, because the crucial factor that affects a woman's decision to delay childbearing is the relative expected utility from work compared to the expected utility from childbearing immediately. The learning effect will encourage delay, and especially so when the probability of learning is high, because a woman's utility from delay is increasing in her expected utility from work in the second period.

Second, consider a change in fertility preferences. Suppose that with probability q, V falls to $v < V$ in the second period with $v < 0$, so that this woman never wishes to have a child. This has the same implications as a rise in income from y to Y, with delay leading to more childless women because some experience a reduction in the utility from childbearing relative to the utility from working. Third, we can model fecundity similarly to child mortality, as a determinant of the success rate of attempted pregnancy. However, the important difference from child mortality is that the success rate declines in the second period because the woman is older. Let us assume that fecundity declines deterministically for all women, and the probability of a pregnancy failing due to

reduced fecundity is θ. Then, the probability of a successful pregnancy in the first period is $(1-\lambda)$ while the same probability in the second period is $(1-\lambda)(1-\theta)$. This means that any woman who delays has a higher probability of a failed pregnancy in the second period, relative to the first period. This raises the proportion of childless women among those women who delay.

D.4 Associations of Income and Education with Childlessness

An interesting question is whether switching is more likely to occur among women with high potential income or low potential income. Income is likely to be correlated with education so this provides an indication of how responses to sulfa exposure may vary with education. The predictions of the model in this regard are ambiguous. Indeed, it is clear from the figure that women with low Y are switchers if they have a low utility V from childbearing, and women with high Y are switchers if they have high V. Thus, the relationship between switchers and income is determined by the joint distribution of Y and V in the population. For example, we would expect increased delay for all income levels if Y and V are positively correlated; if they are strictly negatively correlated, switchers will only be observed for intermediate income levels.

We can also extend the model to take into account a non-constant probability of promotion, p. In particular, if we allow p to depend on Y, then the slope of the solid (indifference) line in the figure will depend on whether the derivative of $p(Y)$ with respect to Y is positive or negative. If the derivative is positive, then the indifference line will be flatter, and there are more women in the population who delay. This is because the return to delay has increased for a given Y, as the probability of achieving the promotion in the second period is higher. In contrast, if p is decreasing with Y (a less realistic assumption), then the indifference line will be steeper.[60]

E Heterogeneity of Main Effects by Race and Education

In this Section we discuss the heterogeneity of our main estimated effects with respect to race and education. Bhalotra and Venkataramani (2012) find that the positive effects of sulfa drugs on the human capital of children born in the antibiotics era were focused among white children, and explain this by arguing that the returns to human capital for blacks were limited due to institutional constraints. The decision to delay fertility and enter the labor market will be affected by constraints to workforce participation and promotion, as well as the relative return of working and waiting,

[60] In Aaronson, Lange, and Mazumder (2014), just as in a classical Beckerian model, more educated women are more likely to be childless because they face a higher opportunity cost of time. Baudin, de la Croix, and Gobbi (2015) describe the same positive association of childlessness with education for the same underlying reason, and additionally assert an association of low levels of education with childlessness, the mechanism being that poverty reduces marriage chances, so that childlessness is U-shaped in education. In contrast to these studies, DeCicca and Krashinsky (2017) adopt an empirical approach similar to that of Aaronson, Lange, and Mazumder (2014) but find that increases in education are associated with lower childlessness, and they argue that this is because educated women are more likely to marry, a similar argument to that invoked by Baudin, de la Croix, and Gobbi (2015) when discussing movements from the bottom to the middle of the education distribution. Our model essentially predicts that child mortality decline can lead to increased childlessness through fertility delay, and education is not a critical pathway.

compared to not working and having children immediately. These constraints and relative returns are likely to be affected by education and race.

Hazard model We explore heterogeneity of the birth timing coefficients by race and education. Each of the four sub-group coefficients is negative and statistically significant for pneumonia mortality decline, and positive but insignificant for maternal mortality decline. In response to pneumonia mortality decline, we find no statistically significant difference in fertility delay in the survival model between the college educated and high school dropouts (Table A.6). We find stronger impacts for blacks than for whites, which can be explained by blacks being exposed to higher mortality rates (typically twice as high) before the introduction of sulfa drugs and thus experiencing larger declines in mortality rates (see Bhalotra and Venkataramani 2012).

Stock model: Childbearing age Fertility responses to pneumonia mortality are statistically significant for white and black women and the coefficients are not significantly different from each other (Table A.7). Responses to maternal mortality are significantly larger for black women, for whom we now see both intensive and extensive margin increases in fertility. For white women, we see only an extensive margin increase, and of a smaller magnitude. Black women were exposed to higher maternal mortality rates pre-sulfa, and may have thus have benefited more from sulfa drugs in combination with medical intervention in childbirth (see Thomasson and Treber 2008 for a discussion of the effect of medical intervention at childbirth during the sulfa era).

We also examined education gradients in impacts of the sulfa innovation, polarizing the educational distribution into those with some college and those who had not completed high school. The fertility response to pneumonia decline for women with college is not significantly different from the response for women who dropped out of high school. However, college-educated women drive the intensive margin decline and high school dropouts drive the extensive margin decline. In fact the increase in childlessness is only statistically significant for high school dropouts (Table A.8).

Stock model: Completed fertility Using completed fertility, we find no statistically significant differences in coefficients by race (Table A.7) or education (Table A.8, both showing estimates for gross fertility).

The responses to pneumonia (child) and maternal mortality decline are larger (though not significantly larger) among black women on both margins, consistent with the pre-sulfa burden of mortality being more than twice as large in the black population. Turning to education gradients, responses to pneumonia tend to be higher among women who are high school dropouts. An exception is that the childlessness coefficient for pneumonia decline is almost identical between high school dropouts and college-educated women, albeit only statistically significant at conventional levels for high school dropouts. In contrast, responses to maternal mortality decline are somewhat larger among the college-educated (though not significantly different). The only coefficients that are statistically significant in response to maternal mortality decline are total fertility and childlessness among college-educated women. This is consistent with women with college having a higher

opportunity cost of childbearing (Baudin, de la Croix, and Gobbi 2015), and also lines up with a similar result in Albanesi and Olivetti (2014).

Labor market outcomes We investigated impacts of sulfa exposure on labor market choices of women, dividing the sample by race and education (Tables A.9 and A.10). The impacts of pneumonia decline were not significantly different by education. However, impacts on work participation, hours and occupational quality were only statistically significant for white women. The fact that the impacts are not significantly different by education is instructive for two reasons. First, interpreted in light of our theoretical model, these results suggest a positive correlation between Y (income) and V (utility from childbearing). Second, it suggests that the labor market impacts of sulfa drugs were not driven by WW2 mobilisation, which only affected the labor force participation of women at the upper end of the education distribution (Goldin and Olivetti 2013). Impacts of maternal mortality decline were not significantly different by race or education.

Marriage market outcomes We uncover a positive, significant effect of the decline in pneumonia mortality on age at first marriage among white women, suggesting that these women delayed marriage as well as childbearing (Table A.11). We also estimate a negative significant effect of the decline in maternal mortality on age at first marriage among white women, consistent with the positive effect of this decline in mortality on the probability of birth after 1937. The marriage market effects are concentrated among the low education group (Table A.12).

Table A.6: Hazard model: Heterogeneous effects by race and education

	(1) - White	(2) - Black	(3) - College	(4) - HS Dropout
			Birth	
$prePneumonia * post1937$	-0.0177**	-0.0551***	-0.0235***	-0.0266**
	(0.0090)	(0.0099)	(0.0063)	(0.0109)
$preMMR * post1937$	0.0023	0.0026	0.0014	0.0034
	(0.0021)	(0.0021)	(0.0015)	(0.0027)
N	4021342	461423	286975	3594323

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia*post1937$ and $preMMR*post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. The first column restricts the sample to whites, the second to blacks, the third to those with some college and the fourth to high school dropouts. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1890-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.7: Fertility in the stock model by race

	Net Fertility			Gross Fertility		
	(1) # Children	(2) # Children | Children >0	(3) Childless	(4) # Children	(5) # Children | Children >0	(6) Childless
Panel A. Whites						
$prePneumonia * Sulfayears$	-0.0403**	-0.0318**	0.0078***	-0.0132	-0.0129	0.0016
	(0.0156)	(0.0142)	(0.0028)	(0.0126)	(0.0122)	(0.0010)
$preMMR * Sulfayears$	0.0027	0.0006	-0.0007*	-0.0040	-0.0009	-0.0000
	(0.0027)	(0.0029)	(0.0004)	(0.0023)	(0.0023)	(0.0001)
N	438414	280761	438414	472851	386482	472851
Panel B. Blacks						
$prePneumonia * Sulfayears$	-0.0743***	-0.0492*	0.0128***	-0.0328**	-0.0164	0.0032
	(0.0200)	(0.0254)	(0.0035)	(0.0130)	(0.0150)	(0.0021)
$preMMR * Sulfayears$	0.0109***	0.0106***	-0.0013**	-0.0009	-0.00274	-0.0002
	(0.0030)	(0.0039)	(0.0006)	(0.0023)	(0.0028)	(0.0004)
N	53749	31714	53749	43470	33271	43470

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). Panel A restricts the sample to whites only, and Panel B restricts the sample to blacks only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.8: Fertility in the stock model by education

	Net Fertility			Gross Fertility		
	(1) # Children	(2) # Children | Children >0	(3) Childless	(4) # Children	(5) # Children | Children >0	(6) Childless
Panel A. At Least Some College						
$prePneumonia * Sulfayears$	-0.0469***	-0.0620***	0.0020	-0.0262**	-0.0241**	0.0023
	(0.0134)	(0.0130)	(0.0052)	(0.0107)	(0.0113)	(0.0019)
$preMMR * Sulfayears$	0.0063**	0.0061**	-0.0007	0.0028*	-0.0001	-0.0009**
	(0.0031)	(0.0027)	(0.0011)	(0.0015)	(0.0019)	(0.0004)
N	36204	20733	36204	74577	56158	74577
Panel B. High School Dropouts						
$prePneumonia * Sulfayears$	-0.0584***	-0.0413***	0.0102***	-0.0364**	-0.0337**	0.0024*
	(0.0136)	(0.0129)	(0.0022)	(0.0160)	(0.0150)	(0.0013)
$preMMR * Sulfayears$	0.0042	0.0011	-0.0011**	0.0006	0.0002	0.0001
	(0.0028)	(0.0027)	(0.0004)	(0.0025)	(0.0024)	(0.0002)
N	295665	217949	295665	292285	241372	292285

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). Panel A restricts the sample to those with some college only, and Panel B restricts the sample to those who did not complete high school. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.9: Labor market outcomes as a function of sulfa exposure by race

	(1) Working	(2) In Labor Force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
Panel A. Whites					
$prePneumonia * sulfayears$	0.0057***	0.0054***	0.1707*	4.8582	0.2077**
	(0.0018)	(0.0018)	(0.0853)	(16.1432)	(0.0676)
$preMMR * sulfayears$	-0.0005*	-0.0005*	0.0067	0.5119	-0.0146
	(0.0003)	(0.0003)	(0.0137)	(2.9529)	(0.0107)
N	649136	649136	462266	277065	649136
Panel B. Blacks					
$prePneumonia * sulfayears$	-0.0021	-0.0010	0.0945	52.3038**	0.1165
	(0.0031)	(0.0032)	(0.1568)	(22.0283)	(0.1411)
$preMMR * sulfayears$	-0.0013**	-0.0012**	0.0034	5.6628*	-0.0840***
	(0.0006)	(0.0006)	(0.0264)	(2.8950)	(0.0215)
N	75014	75014	53061	27762	75014

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where intervalled data is converted to a continuous measure using the midpoints of the intervals. Panel A restricts the sample to whites only, and Panel B restricts the sample to blacks only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 (1900-1931 for column 5) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.10: Labor market outcomes as a function of sulfa exposure by education

	(1) Working	(2) In Labor Force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
Panel A. At Least Some College					
$prePneumonia * sulfayears$	0.0066*	0.0070**	0.3603	41.2755	0.2387*
	(0.0033)	(0.0033)	(0.2924)	(31.0808)	(0.1133)
$preMMR * sulfayears$	-0.0000	-0.0001	0.0027	-4.0435	0.0131
	(0.0006)	(0.0007)	(0.0518)	(7.1769)	(0.0161)
N	61894	61894	43375	43343	66641
Panel B. High School Dropouts					
$prePneumonia * sulfayears$	0.0051**	0.0044**	0.1609*	14.0886	0.3073**
	(0.0021)	(0.0022)	(0.0862)	(15.3356)	(0.1004)
$preMMR * sulfayears$	-0.0006*	-0.0007*	-0.0026	-1.9909	-0.0434**
	(0.0004)	(0.0004)	(0.0151)	(2.4924)	(0.0161
N	443874	443874	330023	142950	494514

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where we convert intervalled data to a continuous measure using the midpoint of each interval. Panel A restricts the sample to those with at least some college, and Panel B restricts the sample to those who did not complete high school only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 (1900-1931 for column 5) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.11: Marriage market outcomes as a function of sulfa exposure by race

	(1) Currently Married	(2) Ever Married	(3) Age at 1st marriage
Panel A. Whites			
$prePneumonia * sulfayears$	-0.0041** (0.0018)	-0.0033** (0.0015)	0.0434* (0.0249)
$preMMR * sulfayears$	0.0011*** (0.0003)	0.0008*** (0.0002)	-0.0052 (0.0052)
N	438414	649136	104643
Panel B. Blacks			
$prePneumonia * sulfayears$	-0.0029 (0.0049)	-0.0058** (0.0026)	-0.1698*** (0.0520)
$preMMR * sulfayears$	-0.0013* (0.0007)	-0.0001 (0.0003)	-0.0044 (0.0086)
N	53749	75014	11477

The dependent variables are: (1) a dummy variable equal to one equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage, only defined for ever married women. Panel A restricts the sample to whites only, and Panel B restricts the sample to blacks only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and influenza and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.12: Marriage market outcomes as a function of sulfa exposure, by education

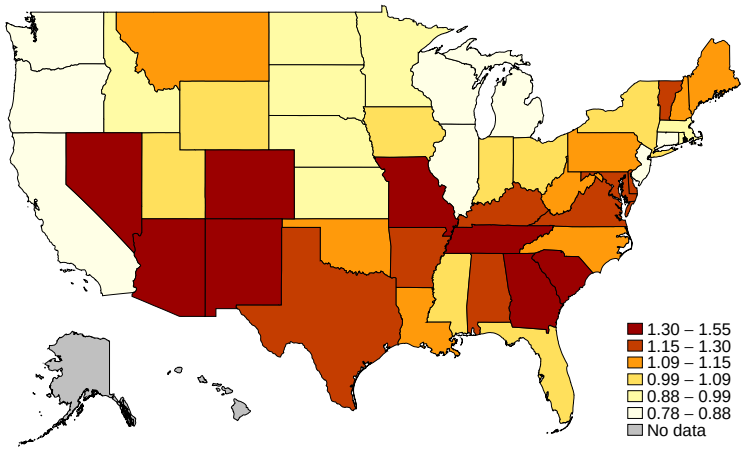
	(1) Currently Married	(2) Ever Married	(3) Age at 1st marriage
Panel A. At Least Some College			
$prePneumonia * sulfayears$	0.0006	0.0005	-0.0341
	(0.0039)	(0.0031)	(0.0570)
$preMMR * sulfayears$	-0.0001	0.0001	-0.0022
	(0.0009)	(0.0006)	(0.0121)
N	36204	61894	14587
Panel B. High School Dropouts			
$prePneumonia * sulfayears$	-0.0013	-0.0018*	0.0157
	(0.0014)	(0.0010)	(0.0289)
$preMMR * sulfayears$	0.0006*	0.0004**	0.0045
	(0.0003)	(0.0002)	(0.0055)
N	295665	443874	56323

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage, only defined for ever married women. Panel A restricts the sample to those with at least some college, and Panel B restricts the sample to those who did not complete high school only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

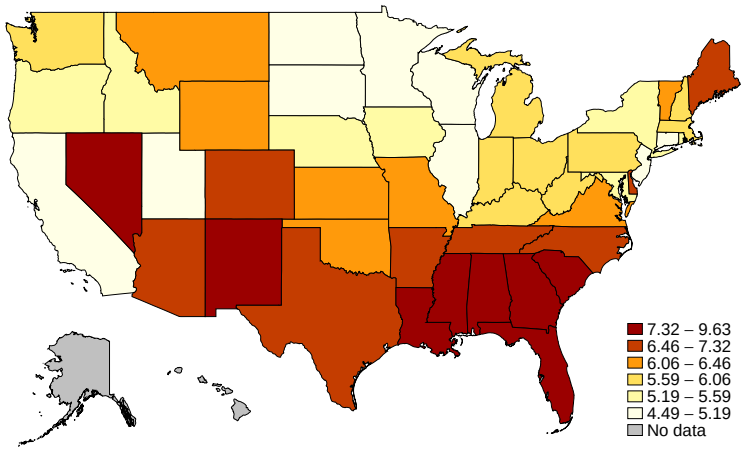
F Additional Results Tables, Figures and Robustness Checks

F.1 Tables and Figures Referred to in the Main Text

Dispersion of mortality rates



(a) Pneumonia Mortality



(b) Maternal Mortality

Figure A.2: Maps showing pneumonia and maternal mortality across the U.S.

This figure displays the average state-level mortality rates between 1930-36.

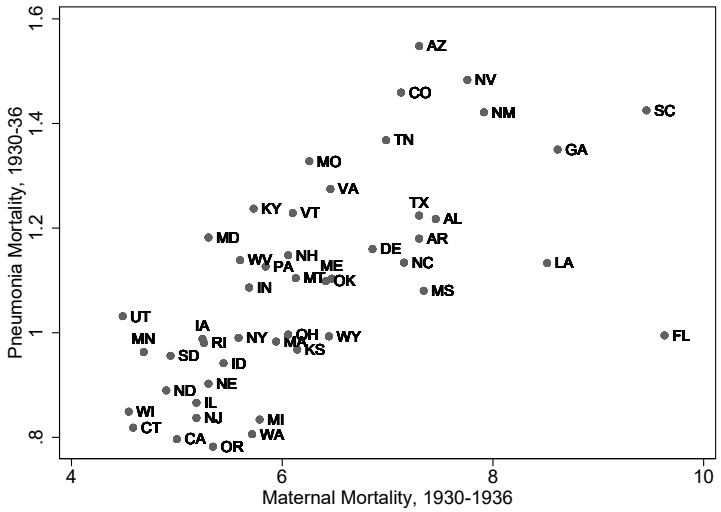


Figure A.3: Pneumonia and Maternal Mortality, United States, 1930-1936

This figure shows the relationship between the average pneumonia and maternal mortality rates in 1930-1936 across different states in the United States. Source: Vital Statistics.

Sensitivity to controls

Table A.13: Net total fertility as a function of sulfa exposure: Sensitivity to controls

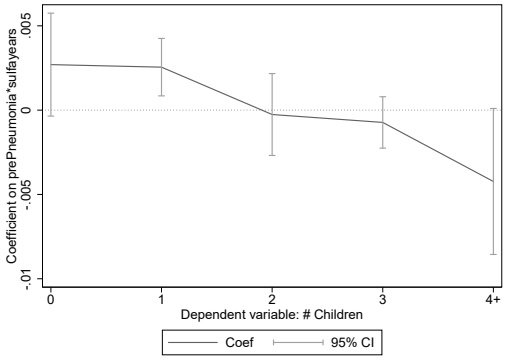
	(1) # Children	(2) # Children
$prePneumonia * sulfayears$	-0.0782***	-0.0483***
	(0.0213)	(0.0127)
$preMMR * sulfayears$	0.0008	0.0035
	(0.0028)	(0.0024)
N	496783	494437
Mean	1.6590	1.6590
Controls		
Baseline controls	Y	Y
State characteristics	N	Y

The dependent variable is the total number of own children living in the household (net fertility). $prePneumonia*sulfayears$ and $preMMR*sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects. The following variables are added in the second column: state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s interacted with $sulfayears$, income and public services, literacy, female labor force participation, and the year of state birth and death registration interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

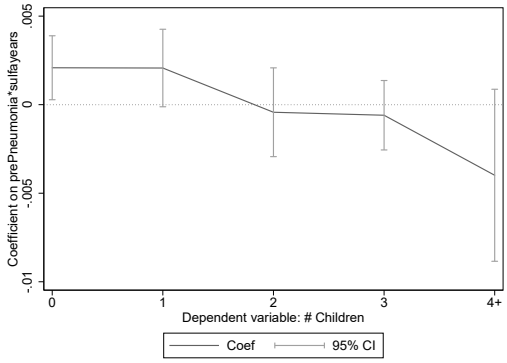
Fertility distribution estimates for completed fertility

Figure A.4: Mortality Reductions and Completed Fertility

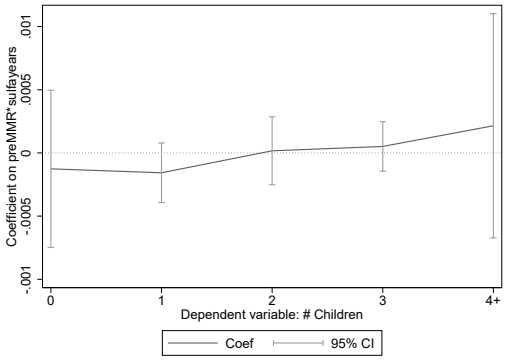
(a) Impact of Pneumonia Mortality Reduction on Distribution of Completed Net Fertility



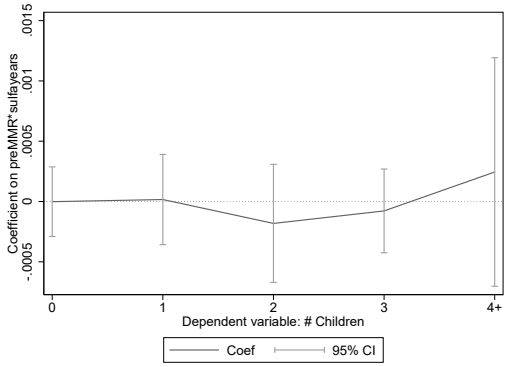
(b) Impact of Pneumonia Mortality Reduction on Distribution of Completed Gross Fertility



(c) Impact of Maternal Mortality Reduction on Distribution of Completed Net Fertility



(d) Impact of Maternal Mortality Reduction on Distribution of Completed Gross Fertility

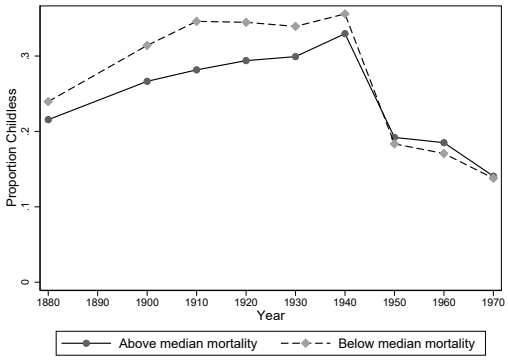


These figures show the coefficient estimates on $prePneumonia * sulfayears$ (Figures (a) and (b)) and $preMMR * sulfayears$ (Figures (c) and (d)), in five separate regressions capturing the impact of mortality reductions on the fertility distribution (having 0, 1, 2, 3, or 4+ children). In all cases these are measures of completed fertility. Figures (a) and (c) show the impact on net completed fertility among woman aged 40-50 at census, and Figures (b) and (d) show the impact on gross completed fertility among women aged 40+ at census.

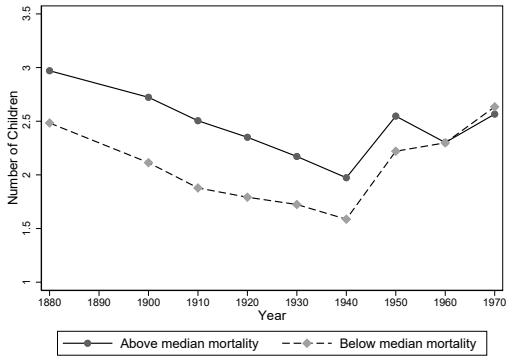
Nonparametric patterns

Figure A.5: Nonparametric event studies of outcomes by above/below median pneumonia mortality

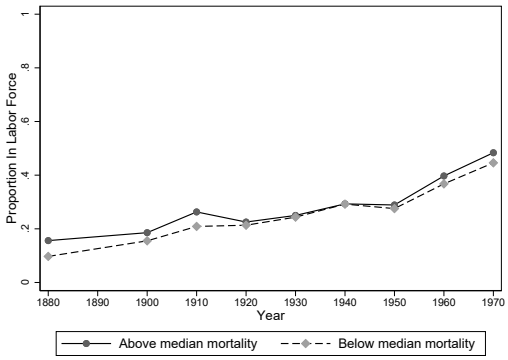
(a) Childlessness



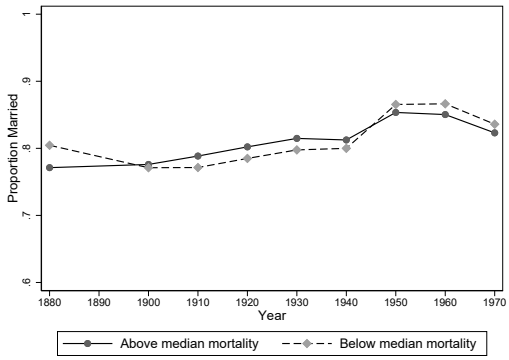
(b) Number of children



(c) Labor force participation



(d) Marital status



These figures show the average state-level outcomes of women in above and below median pneumonia mortality states in each census year, based on average mortality rates in 1930-36. The sample includes all women aged 30-40 at the time of the census.

Distinguishing labor market responses to child pneumonia mortality and adult pneumonia mortality decline

Table A.14: Labor market outcomes as a function of sulfa exposure, comparing child and adult pneumonia mortality

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
$prePneumoniaU5 * sulfayears$	0.0004***	0.0004***	0.0150**	0.0753	0.0142***
magnitude of effect	2.38pp	2.38pp	0.89	4.48	0.84
	(0.0001)	(0.0001)	(0.0062)	(1.0643)	(0.0046)
$prePneumonia25to34 * sulfayears$	-0.0027	-0.0032	0.0895	9.7064	-0.0741
magnitude of effect	-0.64pp	-0.76pp	0.21	23.09	-0.18
	(0.0037)	(0.0039)	(0.1567)	(32.6101)	0.1474)
$preMMR * sulfayears$	-0.0005	-0.0006	-0.0018	-0.0401	-0.0226
magnitude of effect	-1.68pp	-2.02pp	-0.06	-1.35	-0.76
	(0.0003)	(0.0004)	(0.0133)	(2.7790)	(0.0138)
N	727398	727398	517857	306280	727398
Mean	0.3510	0.3710	14.4093	1505.191	12.8097

The magnitudes of effects are calculated as the coefficient x inter-quartile range of mortality variable x average sulfa years in this sample (18.3). The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation, available for the 1950+ censuses; (4) the US Dollar amount of personal earnings in the past year, available for the 1950+ censuses; (5) hours worked in the past week, converted from intervalled data to a continuous measure using the midpoint of each interval. $prePneumoniaU5 * sulfayears$, $prePneumonia25to34 * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia among under 5s, pneumonia among 25-34 year olds and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Descriptive estimates for the joint probability of childlessness and labor force participation

Table A.15: Descriptive regression of relationship between childlessness and labor and marriage market outcomes

	(1)	(2)
	Childless	
	Childbearing women	Completed fertility women
In labor force	0.1916***	0.1121***
	(0.0088)	(0.0037)
Married	-0.4827***	-0.3150***
	(0.0065)	(0.0174)
N	506337	266658
Mean	0.36	0.31

The dependent variable equals one if the woman is childless (based on net fertility) and zero otherwise. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility, labor and marriage market outcomes of women aged 18 to 40 (40 to 50 in column (2)) at the time of the census, aged 6 to 44 in 1937, born in the United States and resident in their birth state at census. The cohorts in this table were born in the years 1893 to 1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, census year and race fixed effects. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Results tables and figures for robustness checks discussed in Section 6.6

Figure A.6: Articles appearing in the New York Times when sulfa drugs arrived to the U.S.

NEW DRUG SAID TO AID IN PUERPERAL FEVER; British Doctors Report Prompt Drop in Temperature and Remission of Symptoms.

Special Cable to THE NEW YORK TIMES. ();
June 06, 1936,
, Section , Page 7, Column , words

PERMISSIONS

[DISPLAYING ABSTRACT]

LONDON, June 5. -- Experiments here with the new drug commonly called prontosil, a German aniline compound, in cases of childbed fever have given exceptional results.

YOUNG ROOSEVELT SAVED BY NEW DRUG; Doctor Used Prontylin in Fight on Streptococcus Infection of the Throat. CONDITION ONCE SERIOUS But Youth, in Boston Hospital, Gains Steadily -- Fiancee, Reassured, Leaves Bedside. YOUNG ROOSEVELT SAVED BY NEW DRUG

Special to THE NEW YORK TIMES. ();
December 17, 1936,
, Section , Page 1, Column , words

PERMISSIONS

[DISPLAYING ABSTRACT]

BOSTON, Dec. 16. -- Franklin D. Roosevelt Jr. faced death from a throat infection last week, it was disclosed tonight by his personal physician, Dr. George Loring Tobey Jr., at the Phillips House of the Massachusetts General Hospital, where young Roosevelt is a patient.

Table A.16: Probability of birth as a function of sulfa exposure - further robustness checks

	(1) Age<10	(2) Conception year	(3) Excl mountain	(4) Excl deep south	(5) Under5s	(6) 2SLS Under5s
			Birth			
$prePneumonia * post1937$	-0.0161**	-0.0263***	-0.0299**	-0.0065		
	(0.0078)	(0.0101)	(0.0129)	(0.0058)		
$preMMR * post1937$	0.0021	0.0029	0.0052*	0.0016	0.0020	0.0056
	(0.0019)	(0.0025)	(0.0028)	(0.0010)	(0.0021)	(0.0046)
$prePneumoniaU5 * post1937$					-0.0006	-0.0039*
					(0.0008)	(0.0023)
N	2810044	4035785	4414170	3941672	4499588	4499792

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic (columns 1-5) and 2SLS (column 6) regressions with standard errors (in parentheses) clustered at the state of birth level. For comparison, the coefficient on $prePneumoniaU5 * sulfayears$ when estimating column (6) using OLS is -0.0008 (s.e. 0.0010). The robustness check in each column is described in detail in Section 6.6. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1890-1928 and are drawn from the 1940 and 1950 US decennial population censuses (for column (1), only potential births between 1940-43 are included from the 1950 census). Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.17: Fertility outcomes as a function of sulfa exposure - further robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel E (Placebo)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.2112	-0.3751	-0.0170			
	(0.2053)	(0.2319)	(0.0323)			
$preMMR * sulfayears$	-0.0080	0.0604	0.0153			
	(0.0449)	(0.0493)	(0.0063)			
N	61918	42447	61918			
Panel F (Excl. 10 and older)						
$prePneumonia * sulfayears$	-0.0227**	-0.0174**	0.0074**			
	(0.0085)	(0.0078)	(0.0029)			
$preMMR * sulfayears$	0.0012	-0.0003	-0.0005			
	(0.0017)	(0.0016	(0.0005)			
N	494437	236499	494437			
Panel G (Excl Mountain West)						
$prePneumonia * sulfayears$	-0.0.0417**	-0.0329**	0.0072***	-0.0225	-0.0196	0.0016
	(0.0155)	(0.0138)	(0.0026)	(0.0156)	(0.0142)	(0.0011)
$preMMR * sulfayears$	0.0042	0.0014	-0.0010**	0.0019	0.0013	-0.00001
	(0.0029)	(0.0030)	(0.0004)	(0.0028)	(0.0026)	(0.0002)
N	483852	306588	483852	509656	413931	509656
Panel H (Excl Deep South)						
$prePneumonia * sulfayears$	-0.0395**	-0.0311**	0.0077**	-0.0104	-0.0107	0.0016
	(0.0157)	(0.0134)	(0.0030)	(0.0130)	(0.0121)	(0.0011)
$preMMR * sulfayears$ 0.0015	-0.0021	-0.0008*	-0.0022	-0.0024	0.0001	
	(0.0023)	(0.0022)	(0.0004)	(0.0022)	(0.0022)	(0.0002)
N	435017	274490	435017	464042	376270	464042

See notes to Table 2 for definitions of outcomes. The robustness checks are described in Section 6.6. For Panel E, our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1897, with outcomes drawn from the 1910-1930 censuses. For other panels, our dataset is a cross-section of outcomes of women aged 6-44 in 1937 and 18-40 at census (columns 1-3) or at least 40 (columns 4-6), born in the U.S. and resident in their birth state at census. The cohorts in this table were born in 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940-1970 US decennial population censuses. See notes to Table A.18 for details on estimation and control variables. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.18: Labor market outcomes as a function of sulfa exposure - further robustness checks

Panel E (Placebo)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0102	0.0211			
	(0.0388)	(0.0435)			
$preMMR * sulfayears$	0.0057	0.0068			
	(0.0083)	(0.0095)			
N	54852	54842			
Panel G (Excl Mountain West)					
$prePneumonia * sulfayears$	0.0065***	0.0062***	0.2163***	35.0206***	0.2744***
	(0.0014)	(0.0014)	(0.0538)	(12.7874)	(0.0460)
$preMMR * sulfayears$	-0.0012***	-0.0012***	-0.0147	-2.1314	-0.0499***
	(0.0002)	(0.0002)	(0.0099)	(2.1562)	(0.0069)
N	712693	712693	507062	300013	712693
Panel H (Excl Deep South)					
$prePneumonia * sulfayears$	0.0056***	0.0052***	0.1668	-15.0912	0.2047***
	(0.0018)	(0.0018)	(0.1032)	(16.5964)	(0.0683)
$preMMR * sulfayears$	-0.0004	-0.0004	0.0065	3.5775	-0.0152
	(0.0003)	(0.0003)	(0.0135)	(2.3918)	(0.0110)
N	642475	642475	459065	274429	642475

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the past week, converted from intervalled data to a continuous variable using the midpoint of each interval. The robustness checks in the different panels are described in Section 6.6. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. For Panel E, our dataset is a cross-section of labor outcomes of women aged 6-44 in 1897, with labor outcomes drawn from the 1910, 1920 and 1930 censuses. Our dataset for panels F-G is a cross-section of labor outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.19: Marriage market outcomes as a function of sulfa exposure - further robustness checks

Panel E (Placebo)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	0.0003	-0.0018	-0.2592***
	(0.0326)	(0.0024)	(0.0112)
$preMMR * sulfayears$	0.0066	-0.0052	-0.0003
	(0.0073)	(0.0005)	(0.0033)
N	61918	135524	7625
Panel G (Excl Mountain West)			
$prePneumonia * sulfayears$	-0.0022	-0.0028**	-0.0274
	(0.0014)	(0.0011)	(0.0177)
$preMMR * sulfayears$	0.0007**	0.0006***	0.0048
	(0.0003)	(0.0002)	(0.0033)
N	483852	904574	302364
Panel H (Excl Deep South)			
$prePneumonia * sulfayears$	-0.0023	-0.0022	-0.0187
	(0.0014)	(0.0013)	(0.0152)
$preMMR * sulfayears$	0.0005**	0.0003*	0.0056**
	(0.0002)	(0.0002)	(0.0026)
N	435017	817174	274187

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage for ever married women. The robustness checks in the different panels are described in Section 6.6. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. The sample includes women aged 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2. For Panel E, our dataset is a cross-section of marriage outcomes of women aged 6-44 in 1897, with outcomes drawn from the 1910, 1920 and 1930 censuses. For Panels F-G, our dataset is a cross-section of marriage outcomes of women aged 6-44 in 1937, born in the United States and resident in their birth state at the time of the census. The cohorts were born in the years 1900-1931 for columns 1 and 3 and 1893-1931 for column 2 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.20: Fertility outcomes as a function of sulfa exposure - further robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel I (Under 5s Pneu)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumoniaU5 * sulfayears$	-0.0025**	-0.0008	0.0006***	-0.0009	-0.0008	0.0001*
	(0.0010)	(0.0009)	(0.0002)	(0.0009)	(0.0008)	(0.0001)
$preMMR * sulfayears$ 0.0019	-0.0011	-0.0007*	-0.0001	-0.0003	0.0000	
	(0.0028)	(0.0026)	(0.0004)	(0.0024)	(0.0023)	(0.0002)
N	494437	313981	494437	518933	421983	518933
Panel J (Under 5s 2SLS)						
$prePneumoniaU5 * sulfayears$	-0.0071***	-0.0050**	0.0013***	-0.0032	-0.0028	0.0003*
	(0.0027)	(0.0024)	(0.0004)	(0.0022)	(0.0019)	(0.0002)
$preMMR * sulfayears$	0.0062	0.0028	-0.0014**	0.0019	0.0014	-0.0001
	(0.0046)	(0.0045)	(0.0006)	(0.0037)	(0.0032)	(0.0003)
N	494437	313981	494437	518933	421983	518933
Panel K (Incl Migrants)						
$prePneumonia * sulfayears$	-0.0253***	-0.0159*	0.0052***	-0.0145	-0.0145	0.0012
	(0.0083)	(0.0087)	(0.0016)	(0.0105)	(0.0095)	(0.0007)
$preMMR * sulfayears$	0.0018	-0.0005	-0.0006**	0.0008	0.0004	-0.0001
	(0.0017)	(0.0019)	(0.0003)	(0.0022)	(0.0021)	(0.0001)
N	828558	548080	828558	955497	780370	955497
Panel L (2SLS Migrants)						
$prePneumonia * sulfayears$	-0.0408***	-0.0300**	0.0080***	-0.0262	-0.0255	0.0023*
	(0.0127)	(0.0141)	(0.0024)	(0.0171)	(0.0158)	(0.0012)
$preMMR * sulfayears$	0.0036	0.0006	-0.0010***	0.0015	0.0007	-0.0002
	(0.0023)	(0.0027)	(0.0004)	(0.0032)	(0.0030)	(0.0002)
N	809989	533144	809989	880951	718855	880951

See notes to Table 2 for definitions of outcomes. The robustness checks are described in Section 6.6. Our dataset is a cross-section of outcomes of women aged 6-44 in 1937 and 18-40 at census (columns 1-3) or at least 40 (columns 4-6), born in the U.S. and, in Panels I and J, resident in their birth state at census, while in Panels K and L, resident in any state. The cohorts in this table were born in 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940-1970 US decennial population censuses. See notes to Table A.18 for details on estimation and control variables. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.21: Labor market outcomes as a function of sulfa exposure - further robustness checks

Panel I (Under 5s Pneu)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumoniaU5 * sulfayears$	0.0003***	0.0003**	0.0163***	0.2278	0.0131***
	(0.0001)	(0.0001)	(0.0051)	(0.9473)	(0.0043)
$preMMR * sulfayears$	-0.0006*	-0.0007**	0.0011	0.2777	-0.0250**
	(0.0003)	(0.0003)	(0.0120)	(2.5478)	(0.0119)
N	727398	727398	517857	306280	727398
Panel J (Under 5s 2SLS)					
$prePneumoniaU5 * sulfayears$	0.0009***	0.0008**	0.0290**	1.0635	0.0365***
	(0.0003)	(0.0003)	(0.0125)	(2.2722)	(0.0136)
$preMMR * sulfayears$	-0.0011**	-0.0011**	-0.0109	-0.4720	-0.0468**
	(0.0006)	(0.0005)	(0.0194)	(3.3553)	(0.0230)
N	727398	727398	517857	306280	727398
Panel K (Incl Migrants)					
$prePneumonia * sulfayears$	0.0033***	0.0030**	0.1496**	-21.0175	0.1214***
	(0.0012)	(0.0012)	(0.0693)	(16.5351)	(0.0449)
$preMMR * sulfayears$	-0.0007***	-0.0008***	-0.0141	2.9099	-0.0265***
	(0.0002)	(0.0002)	(0.0114)	(2.8243)	(0.0079)
N	1264807	1264807	980312	673706	1264807
Panel L (2SLS Migrants)					
$prePneumonia * sulfayears$	0.0045**	0.0040**	0.2290**	-34.3413	0.1612**
	(0.0018)	(0.0019)	(0.1049)	(23.9145)	(0.0676)
$preMMR * sulfayears$	-0.0008***	-0.0009***	-0.0201	4.1939	-0.0328***
	(0.0003)	(0.0003)	(0.0150)	(3.5770)	(0.0108)
N	1222295	1222295	939159	633897	1222295

See notes to Table A.18 for details on estimation and control variables. The robustness checks in the different panels are described in Section 6.6. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions in Panels I and K (2SLS, Panels J and L) with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and, for Panels I and J, resident in their birth state at the time of the census, while for Panels K and L, resident in any state. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.22: Marriage market outcomes as a function of sulfa exposure - further robustness checks

Panel I (Under 5s Pneu)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumoniaU5 * sulfayears$	-0.0001*	-0.0002**	0.0009
	(0.0001)	(0.0001)	(0.0021)
$preMMR * sulfayears$	0.0006**	0.0005**	0.0048
	(0.0002)	(0.0002)	(0.0057)
N	494437	727398	116632
Panel J (2SLS)			
$prePneumoniaU5 * sulfayears$	-0.0003*	-0.0005**	0.0003
	(0.0002)	(0.0002)	(0.0035)
$preMMR * sulfayears$	0.0007***	0.0008**	0.0052
	(0.0003)	(0.0003)	(0.0061)
N	494437	727398	116632
Panel K (Incl Migrants)			
$prePneumonia * sulfayears$	-0.0004	-0.0009*	-0.0132
	(0.0009)	(0.0005)	(0.0132)
$preMMR * sulfayears$	0.0003*	0.0001	0.0039*
	(0.0002)	(0.0001)	(0.0021)
N	828558	1592612	595742
Panel L (2SLS Migrants)			
$prePneumonia * sulfayears$	-0.0008	-0.0017**	-0.0214
	(0.0014)	(0.0008)	(0.0219)
$preMMR * sulfayears$	0.0005**	0.0003*	0.0054*
	(0.0002)	(0.0001)	(0.0030)
N	809989	1527667	587994

See notes to Table A.19 for details on estimation and control variables. The robustness checks in the different panels are described in Section 6.6. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions in Panels I and K (2SLS, Panels J and L) with standard errors (in parentheses) clustered at the state of birth level. The sample includes women aged 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2. Our dataset is a cross-section of marriage outcomes of women aged 6-44 in 1937, born in the United States and, in Panels I and J, resident in their birth state at the time of the census, and in Panels K and L, resident in any state. The cohorts were born in the years 1900-1931 for columns 1 and 3 and 1893-1931 for column 2 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.23: Linear trends in birth probability in above and below median mortality states, 1930-1936

	(1)	(2)	(3)
		Birth	
$AboveMedPneu * trend$	-0.0001	-0.0001	
	(0.0002)	(0.0003)	
$AboveMedMMR * trend$	0.0003		0.0003
	(0.0003)		(0.0003)
N	2230331	2230331	2230331

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. The variable *trend* is a linear time trend. *AboveMedPneu* is a dummy variable that equals one if a state had an above median average value of *prePneumonia* in 1930-1936 and zero otherwise. The variable *AboveMedMMR* has the analogous definition for *preMMR*. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1936, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *post*1937. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.24: Binary difference-in-difference estimates of the effect of sulfa exposure on fertility

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * treated$	-0.8565***	-0.7925**	0.1386**	-0.4930	-0.5562	0.0325
	(0.3830)	(0.3472)	(0.0676)	(0.3667)	(0.3502)	(0.0253)
$preMMR * treated$	0.1022	0.0724	-0.0181*	-0.0164	-0.0105	0.0032
	(0.0643)	(0.0581)	(0.0102)	(0.0567)	(0.0524)	(0.0044)
N	279899	182808	279899	163036	137303	163036

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). *treated* is a variable that equals one if a woman was exposed to sulfa drugs for her entire fertile period, and equals zero if a woman was exposed for no years. Women exposed for only some years are excluded from these regressions. *prePneumonia* and $preMMR$ are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-15 or 40-44 in 1937 and 18-50 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1897 and 1922-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *treated*. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.25: Binary difference-in-difference estimates of the effect of sulfa exposure on labor market outcomes

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * treated$	0.1339**	2.3366***	-3303.3419***	-0.0440	4.5693
	(0.0588)	(0.0597)	(0.5128)	(64.3696)	(2.4842)
$preMMR * treated$	-0.0295***	-0.0335***	2.8876***	338.6701***	-1.2668**
	(0.0102)	(0.0103)	(0.0474)	(17.5327)	(0.4305)
N	279899	279899	245681	168344	279899

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the past week, where we convert the intervalled measure to a continuous measure using the midpoint of each interval. *treated* is a variable that equals one if a woman was exposed to sulfa drugs for her entire fertile period, and equals zero if a woman was exposed for no years. Women exposed for only some years are excluded from these regressions. *prePneumonia* and $preMMR$ are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 6-15 or 40-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1897 and 1922-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *treated*. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.26: Binary difference-in-difference estimates of the effect of sulfa exposure on marriage market outcomes

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * treated$	-0.0440	-0.0998**	0.3031
	(0.0398)	(0.0376)	(0.6092)
$preMMR * treated$	0.0201***	0.0175***	0.2636***
	(0.0062)	(0.0063)	(0.0986)
N	279899	279899	86390

The dependent variables are: (1) a dummy variable equal to one equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage, only defined for ever married women. *treated* is a variable that equals one if a woman was exposed to sulfa drugs for her entire fertile period, and equals zero if a woman was exposed for no years. Women exposed for only some years are excluded from these regressions. *prePneumonia* and *preMMR* are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 6-15 or 40-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1897 and 1922-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *treated*. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.27: Migration as a function of sulfa exposure

	(1) Birth - Migrants	(2) Birth - 2SLS Migrants	(3) Pr(in migrant sample)	(4) Migrated between 1935-40
$prePneumonia * post1937$	-0.0219**	-0.0386*		
	(0.0102)	(0.0211)		
$preMMR * post1937$	0.0015	0.0024		
	(0.0024)	(0.0043)		
$prePneumonia * sulfayears$			0.0020	-0.0045
			(0.0031)	(0.0072)
$preMMR * sulfayears$			-0.0005	0.0001
			(0.0005)	(0.0014)
N	6349665	6319430	1122827	56722

Columns 1 and 2: The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia*post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. Column 1 is a Logistic regression and Column 2 a 2SLS regression. Standard errors (in parentheses) are clustered at the state of birth level. The robustness check in each column is described in detail in Section 6.6. Our dataset for columns 1 and 2 is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in any state at the time of the census. The cohorts in columns 1 and 2 were born in the years 1890-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. Columns 3 and 4: The dependent variable in Column 3 is a dummy variable that equals one if a woman's census state is different from her birth state, and zero otherwise. The dependent variable in Column 4 equals one if a woman reported in the 1940 census that she has migrated in the last 5 years, and equals zero if she reported that she did not migrate. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and at least 40 at the time of the census and born in the United States. The cohorts in this sample were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses (Column 3) and the 1940 US decennial population census (Column 4). Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

F.2 Additional Robustness Checks

Alternative sample definitions First, we show that our stock model results are not sensitive to sample definitions. We reestimate the net fertility results for 18-36 year olds at the time of the census (a child born to a woman aged 18 would leave home at 36, hence this measure minimises underreporting of children who have left home). These results are in Table A.28. All the results are statistically significant and the magnitudes are comparable to those in the main text. Table A.29 complements this analysis by presenting results for gross uncompleted fertility; that is, gross fertility for 18-40 year olds. The coefficients are comparable in magnitude to the main text, although they are not precisely estimated; this is likely driven by the fact that the gross fertility question was only asked to ever married women in the 1940 and 1950 censuses, and 95% of the sample in these regressions comes from these two censuses. As the main results suggest that fertility and marriage decisions are intertwined, restricting the sample to ever married women leads to a select sample of women.

In Table A.30, we show that the labor supply results are robust to using a sample of 18-40 year olds and 18-60 year olds; as with the fertility results, the coefficients have the largest magnitudes for the youngest sample. This Table also shows robustness to widening the marriage market sample. When we widen the sample to include women up to age 60, the coefficient for current marital status is attenuated and insignificant, showing the importance of precise sample definitions.

Outliers Next, we reestimate the main results but excluding New Mexico, which was shown to be an outlier state in Figure 9. The hazard model results are in column (1) of Table A.32, while the stock model results are in Panel I of Tables A.33 (fertility) and A.34-A.35 (labor and marriage markets). The exclusion of New Mexico does not change the results in a substantive way.

OLS and Woman Fixed Effects In order to verify that our results are similar in a simpler estimation model, we estimate the hazard model using OLS (Table A.32). In the same table, to control for time invariant unobserved factors at the woman level that affect birth probability and potentially are also correlated with mortality rates, we estimate the hazard model with woman fixed effects. The coefficients are similar to the main results in Table 1, but they are less precisely estimated.

Multiple hypothesis testing Finally, in Panel L of Table A.34, we adjust the standard errors from the main results (Table 4) for multiple hypothesis testing. (We do not adjust the standard errors for fertility because these variables are all defined based on one originating variable.) In particular, we implement the procedure described in Aker, Boumnijel, McClelland, and Tierney 2014, which adjusts standard errors to take into account correlation between outcomes. The formula

for the adjusted p-values is

$$\begin{aligned} p^{new} &= 1-(1-p^{old})^{A} \\ A &= (1-c)^{\#outcomes}, \end{aligned}$$

where c is the average correlation between all other outcomes in the group. As we only consider two marriage market outcomes, this formula can only be implemented for the labor market outcomes. The adjusted standard errors do not change the significance of the results in a substantive way.

Table A.28: Net fertility as a function of sulfa exposure: 18-36 year old women

	(1) # Children	(2) # Children | Children >0	(3) Childless
$prePneumonia * sulfayears$	-0.0408***	-0.0297**	0.0089***
	(0.0119)	(0.0130)	(0.0029)
$preMMR * sulfayears$	0.0029	0.0010	-0.0008*
	(0.0024)	(0.0028)	(0.0005)
N	393720	234416	393720

The dependent variables are the total number of children (column 1), the total number of children conditional on having at least one (column 2) and a dummy variable that equals one if the woman has zero children and zero otherwise (column 3), all based on net fertility (the number of own children living in the household). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-36 at census enumeration, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years1904-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.29: Gross fertility as a function of sulfa exposure: 18 to 40 year old women

	(1) # Children	(2) # Children | Children >0	(3) Childless
$prePneumonia * sulfayears$	-0.0288	-0.0275	0.0029
	(0.0201)	(0.0205)	(0.0029)
$preMMR * sulfayears$	0.00003	0.00002	-0.0000
	(0.00004)	(0.00005)	(0.0000)
N	170537	138760	170537
Controls	0.0700	0.0711	0.0381

The dependent variables are the total number of children (column 1), the total number of children conditional on having at least one (column 2) and a dummy variable that equals one if the woman has zero children and zero otherwise (column 3), all defined by gross fertility (the number of live births a woman ever had). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.30: Labor market outcomes as a function of sulfa exposure - alternative samples

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
Panel A			18 to 40 year old women		
$prePneumonia * sulfayears$	0.0074**	0.0064*	0.2727*	-5.3710	0.3561***
	(0.0025)	(0.0026)	(0.1351)	(12.0006)	(0.0878)
$preMMR * sulfayears$	-0.0012**	-0.0012**	-0.0291	-2.7448	-0.0525***
	(0.0004)	(0.0004)	(0.0207)	(1.7008)	(0.0145)
N	494437	494437	317867	152468	494437
Panel B			18 to 60 year old women		
$prePneumonia * sulfayears$	0.0043**	0.0040**	0.1481*	-9.0074	0.1775**
	(0.0015)	(0.0015)	(0.0570)	(11.9414)	(0.0518)
$preMMR * sulfayears$	-0.0005*	-0.0005*	0.0030	-0.1509	-0.0251**
	(0.0002)	(0.0002)	(0.0100)	(2.6067)	(0.0084)
N	922769	922769	713228	482383	922769

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where intervalled data is converted to a continuous measure using the midpoint of each interval. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 6-44 in 1937, born in the United States and resident in their birth state at the time of the census, with age at census restrictions shown above the relevant columns in the table. The cohorts in this table were born in the years 1900-1931 (Panel A) or 1893-1931 (Panel B) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.31: Marriage market outcomes as a function of sulfa exposure - alternative samples

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
Panel A		18 to 50 year old women	
$prePneumonia * sulfayears$	-0.0013	-0.0032**	-0.0170
	(0.0011)	(0.0012)	(0.0152)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0033
	(0.0002)	(0.0002)	(0.0022)
N	727398	727398	181562
Panel B		18 to 60 year old women	
$prePneumonia * sulfayears$	-0.0005	-0.0025**	-0.0294*
	(0.0011)	(0.0011)	(0.0155)
$preMMR * sulfayears$	0.0004***	0.0004**	0.0045
	(0.0001)	(0.0002)	(0.0027)
N	922769	922769	309077

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) age at first marriage, only defined for ever married women. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 6-44 in 1937, born in the United States and resident in their birth state at the time of the census, with age at census restrictions shown above the relevant columns in the table. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.32: Probability of birth as a function of sulfa exposure - additional robustness checks

	(1) - Excl. New Mexico	(2) - Pneu	(3) - MMR Birth	(4) - OLS	(5) - OLS+WFE
$prePneumonia * post1937$	-0.0241**	-0.0158*		-0.0253*	-0.0185
	(0.0104)	(0.0084)		(0.0134)	(0.0209)
$preMMR * post1937$	0.0037		0.0014	0.0043	0.0054
	(0.0025)		(0.0023)	(0.0032)	(0.0045)
N	4485763	4499588	4499588	4499792	4499792

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1890-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *post*1937. Column (6) adds woman fixed effects. Columns (5) and (6) are estimated using OLS. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.33: Net and gross fertility as a function of sulfa exposure - additional robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel a (Excl. New Mexico)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0474***	-0.0341***	0.0088***	-0.0207*	-0.0184*	0.0021**
	(0.0124)	(0.0117)	(0.0023)	(0.0119)	(0.0107)	(0.0009)
$preMMR * sulfayears$	0.0040*	0.0011	-0.0010***	0.0011	0.0007	-0.00003
	(0.0023)	(0.0026)	(0.0003)	(0.0023)	(0.0021)	(0.0001)
N	492776	312786	492776	517651	420859	517651
Panel b (Pneu only)						
$prePneumonia * sulfayears$	-0.0397***	-0.0324***	0.0067**	-0.0192*	-0.0179*	0.0021**
	(0.0131)	(0.0097)	(0.0027)	(0.0100)	(0.0093)	(0.0009)
N	494437	313981	494437	518933	421983	518933
Panel c (MMR only)						
$preMMR * sulfayears$	-0.0005	-0.0019	-0.0002	-0.0009	-0.0011	0.0002
	(0.0030)	(0.0025)	(0.0005)	(0.0021)	(0.0020)	(0.0002)
N	494437	313981	494437	518933	421983	518933

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.34: Labor market outcomes as a function of sulfa exposure - additional robustness checks

Panel a (Excl. New Mexico)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1970**	7.9247	0.2419***
	(0.0017)	(0.0018)	(0.0750)	(15.3758)	(0.0634)
$preMMR * sulfayears$	-0.0008***	-0.0008***	-0.0023	-0.0013	-0.0347**
	(0.0003)	(0.0003)	(0.0120)	(2.7073)	(0.0109)
N	725118	725118	516184	305575	725118
Panel b (Pneu only)					
$prePneumonia * sulfayears$	0.0040**	0.0036*	0.2000**	7.0433	0.1642*
	(0.0017)	(0.0018)	(0.0753)	(13.2783)	(0.0662)
N	727398	727398	517857	306280	727398
Panel c (MMR only)					
$preMMR * sulfayears$	-0.0003	-0.0004	0.0166	0.4821	-0.0128
	(0.0003)	(0.0003)	(0.0140)	(2.3500)	(0.0136)
N	727398	727398	517857	306280	727398
Panel d (Mult. Hypothesis)					
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1991**	7.7366	0.2421***
	(0.0018)	(0.0019)	(0.0857)	(54.9474)	(0.0675)
$preMMR * sulfayears$	-0.0008**	-0.0008**	0.0004	-0.1243	-0.0322***
	(0.0003)	(0.0003)	(0.6441)	(234.485)	(0.0121)
N	727398	727398	517857	306451	727398

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the past week, where intervalled data is converted to a continuous measure using the midpoint of each interval. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 6-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.35: Marriage market outcomes as a function of sulfa exposure - additional robustness checks

Panel a (Excl. New Mexico)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0023*	-0.0032**	0.0010
	(0.0012)	(0.0012)	(0.0242)
$preMMR * sulfayears$	0.0007***	0.0006***	0.0048
	(0.0002)	(0.0002)	(0.0057)
N	492776	725118	116261
Panel b (Pneu only)			
$prePneumonia * sulfayears$	-0.0008	-0.0018	0.0154
	(0.0013)	(0.0013)	(0.0203)
N	494437	727398	116632
Panel c (MMR only)			
$preMMR * sulfayears$	0.0004*	0.0003	0.0055
	(0.0002)	(0.0002)	(0.0048)
N	494437	727398	116632
Panel d (Mult. Hypothesis)			
$prePneumonia * sulfayears$	-0.0023	-0.0032**	0.0021
	(0.0016)	(0.0014)	(0.0652)
$preMMR * sulfayears$	0.0006***	0.0006***	0.00053
	(0.0002)	(0.0002)	(0.0066)
N	494437	727398	116632

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at fist marriage, only defined for ever married women. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 6-44 in 1937 and 18-40 at the time of the census for columns 1 and 3, 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

G Sulfa Exposure and Education

The intuition for investment in human capital is similar to that for decisions about work: women may delay fertility to extend their education and then enter the labor market, similar to the mechanism in Goldin and Katz (2002). However, our sample is of women who were of reproductive age when sulfa drugs were introduced, a large fraction of whom had completed their education: these were 74% in the hazard sample and 68% in the completed fertility stock sample. Note that though the children of these women, who were born into an environment with sulfa drugs, did grow up to acquire more education; see Bhalotra and Venkataramani (2012). Restricting the sample to those that had not completed their education (for example, those aged under 21 in 1937), would result in a sample of women with little variation in sulfa exposure, making it more difficult to identify the effect of sulfa drugs on education decisions. In addition, the main estimates are not sensitive to the removal of woman's education fixed effects as a control variable. We nevertheless investigated education acquisition by creating a sample of women aged 15 to 25 in 1937 and defining exposure to sulfa drugs as being aged 20 or under in 1937, as college completion typically occurred in the early 20s. We find no evidence that exposure to sulfa drugs led to higher investment in college, though we do find a 5.4 percentage point increase in the probability of high school completion, relative to a baseline mean of 19.2% for this subsample of women (Table A.36). In contrast, the reduction in maternal mortality reduced high school completion and increased the probability that a woman dropped out of high school, consistent with the effects of sulfa exposure on labor market outcomes.

In our sample, childlessness is higher among college-educated women and high school dropouts than among women in the middle who completed high school. Hence, human capital accumulation does not appear to be the relevant pathway here. To confirm this, we re-estimated the main equations for fertility, the labor market and marriage outcomes, using a sample of women who were at least 21 (so that they had plausibly completed education choices) when sulfa drugs arrived, and the pattern of results is very similar (Tables A.37-A.40).

Table A.36: Education as a function of sulfa exposure - women aged 15-25 in 1937

	(1) Some College	(2) High School graduate	(3) HS dropout	(4) Some College	(5) High School graduate	(6) HS dropout
	Aged 21-40 at census interview			Aged 40 or older at census interview		
$prePneumonia * treated\_educ$	-0.0113	0.0541**	-0.0428	0.0012	0.0502	-0.0514
	(0.0108)	(0.0264)	(0.0265)	(0.0168)	(0.0347)	(0.0318)
$preMMR * treated\_educ$	-0.0009	-0.0093***	0.0101**	-0.0034	-0.0013	0.0047
	(0.0016)	(0.0033)	(0.0041)	(0.0033)	(0.0032)	(0.0042)
N	199161	199161	199161	186067	186067	186067

The dependent variables are dummy variables for the highest level of education achieved by the woman. $treated\_educ$ takes the value one if a woman was 20 or under in 1937, and the value zero if she was 21 or older. The sample is further restricted to woman aged at least 21 at the time of census enumeration so that they had completed their education. $prePneumonia$ and $preMMR$ are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 15-25 in 1937 and 21-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1912-1922 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.37: Probability of birth as a function of sulfa exposure - women aged 21 and over in 1937

	(1) Birth
$prePneumonia * post1937$	-0.0240* (0.0125)
$preMMR * post1937$	0.0007 (0.0030)
N	3306092

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, and aged at least 21 in 1937, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1890-1906 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.38: Net and gross fertility as a function of sulfa exposure - women aged 21 and over in 1937

	(1)	(2)	(3)	(4)	(5)	(6)
	Net Fertility			Gross Fertility		
	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0572***	-0.0478***	0.0074***	-0.0314**	-0.0322**	0.0026*
	(0.0158)	(0.0168)	(0.0024)	(0.0128)	(0.0120)	(0.0016)
$preMMR * sulfayears$	0.0062*	0.0048	-0.0007	0.0026	0.0031	0.0000
	(0.0032)	(0.0033)	(0.0005)	(0.0022)	(0.0020)	(0.0004)
N	183740	124714	183740	316159	247946	316159

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 21-44 in 1937 and 24-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1916-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.39: Labor market outcomes as a function of sulfa exposure - women aged 21 and over in 1937

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0043*	0.0049*	-0.0500	-15.7802	0.1669
	(0.0024)	(0.0025)	(0.0992)	(21.5968)	(0.1061)
$preMMR * sulfayears$	-0.0007	-0.0009*	0.0250	-2.9891	-0.0320*
	(0.0005)	(0.0005)	(0.0242)	(5.0821)	(0.0187)
N	325467	325467	174280	77937	325467

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where intervalled data is converted to a continuous measure using the midpoint of each interval. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 21-44 in 1937 and 24-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1916-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.40: Marriage market outcomes as a function of sulfa exposure - women aged 21 and over in 1937

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0048*	-0.0026	0.0650
	(0.0025)	(0.0017)	(0.0462)
$preMMR * sulfayears$	0.0008	0.0004	-0.0127
	(0.0005)	(0.0003)	(0.0092)
N	183740	325467	27596

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at fist marriage, only defined for ever married women. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 21-44 in 1937 and 24-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1916-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

H Cross-Sectional Relationships between Mortality, Fertility, Labor and Marriage Choices in 1930 and in 2015

In this Appendix we first explain the construction of the correlational figures in the main text, and then discuss further cross-sectional relationships between mortality rates and women's choices over fertility, the labor market and the marriage market. Figure 9 in the main text shows two plots, each of which are a measure of net fertility plotted against pneumonia mortality. Total net fertility is the weighted state-level average of the number of own children living with a woman at the time of the 1930 census, restricted to women aged 25-40 at the time of the census. Net childlessness takes the value of one when this number is zero and a value of zero otherwise. The charts utilise state-level all-age mortality from pneumonia and influenza 1930-36 per 1000 population, which is the treatment variable used in the main analysis of the paper.

In Figure A.7, we display these same correlations for two other measures of child mortality: child mortality among under-1s from all causes, and under-2s mortality due to diarrhea. In Figure A.8, we plot the same cross-sectional correlations, but with mortality from diseases that did not affect infants: heart disease, diabetes and nephritis (liver disease); these measures are all for the year 1930. We observe a *positive* relationship between mortality and childlessness, and a negative relationship between mortality and fertility; this is consistent with less healthy individuals having fewer children and being more likely to have no children, for example due to sterility or poverty (as in Baudin, de la Croix, and Gobbi 2015).[61]

We show cross-sectional relationships between gross fertility and mortality rates in Figures A.9 and A.10. These are similar to Figure A.7, with the issue that the question on gross fertility was only asked in the 1900 and 1910 censuses, and the 1940 and subsequent censuses. As we do not have this information for 1930, we show the data for the closest available pre-sulfa year, namely 1910. An additional complication is that mortality data from the Vital Statistics for this year is missing for more than half of states, which cannot be assumed to be a random sample. Therefore, we plot 1910 gross fertility against 1930 mortality data; this has its obvious concerns and the charts should be interpreted with caution. That being said, we find patterns that are broadly in line with those for 1930 net fertility (Figure A.9). Childlessness is decreasing with child mortality, but increasing with adult mortality (Figure A.10). Total gross fertility follows approximately the opposite pattern, with the exception of nephritis mortality.

Next, we consider an alternative measure of child mortality, that is measured per live births, rather than per 1000 population as in the preceding charts. This is to show that the observed relationship is not a mechanical one between childlessness and child mortality (high childlessness

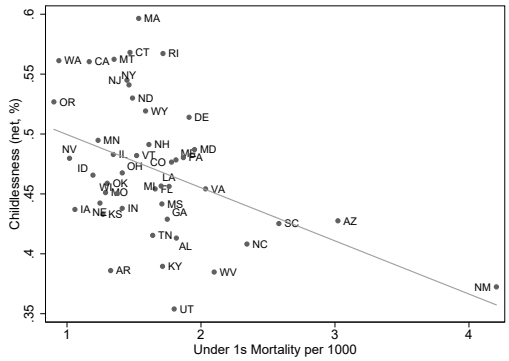
[61] In these figures, the rates of net childlessness are fairly high while the rates of gross childlessness are low; this is because net childlessness overestimates true childlessness due to children that have left home, while gross childlessness underestimates true childlessness due to children that have died during infancy. This is potentially an issue if this over or underestimation is correlated with child mortality; this is plausible for gross fertility but perhaps less plausible for net fertility, which would require either earlier age at childbearing or earlier age of children leaving home in low child mortality areas. Further, the difference-in-difference between net and gross fertility and high and low mortality states is stable between 1900-1910.

implies few births, implying lower child mortality). We plot net fertility in 1930 and gross fertility in 1910 against 1929 infant mortality per 1000 live births extracted from the Vital Statistics 1940 yearbook (Dunn 1943, Figure A.11). The relationships are very similar to those displayed in the earlier charts.

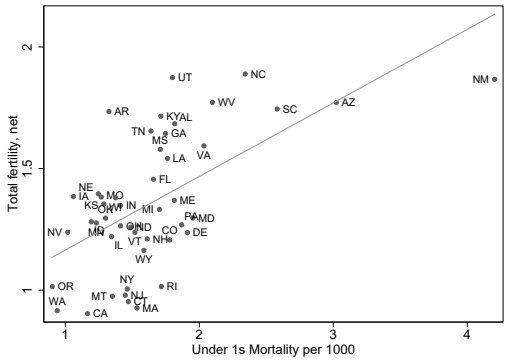
Finally, we consider the cross-sectional relationships between labor market behavior, marriage market behavior and child mortality. To measure labor market behavior, we use the variable In Labor Force, which was available in the 1930 census. For marriage market behavior, we consider the Currently Married question. For both questions, we make the same sample selections as are made in the main analysis of this paper (labor market answers were retained for 18-50 year old women at the time of the census, while currently married answers were retained for 18-40 year old women). Figure A.12 shows the labor market plots for child mortality and adult mortality, Figure A.13 shows these plots for the marriage market, while the comparable plots for infant mortality per 1000 live births are in Figure A.11. Although these Figures will mask many unobservable factors, such as education levels, health status and income, they are surprisingly consistent with the fertility charts. On the whole, working status is negatively correlated with child mortality and positively correlated with adult mortality, although the correlation is at times weak, while marriage rates are positively correlated with child mortality and negative correlated with adult mortality.

Figure A.7: Child Mortality and Net Fertility in 1930 across US states

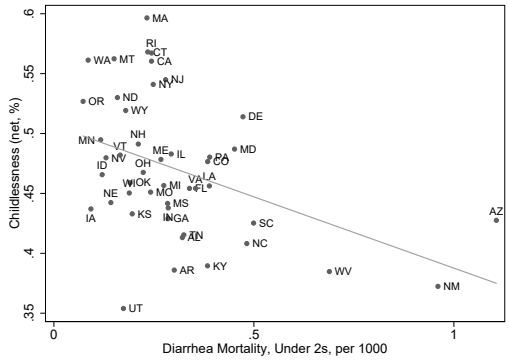
(a) Under 1s Mortality and Net Childessness



(b) Under 1s Mortality and Net Fertility



(c) Diarrhea Under 2s Mortality and Net Childessness



(d) Diarrhea Under 2s Mortality and Net Fertility

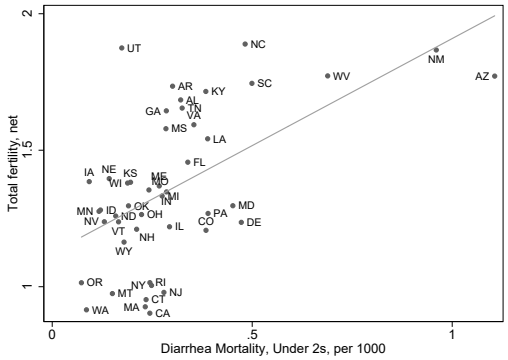
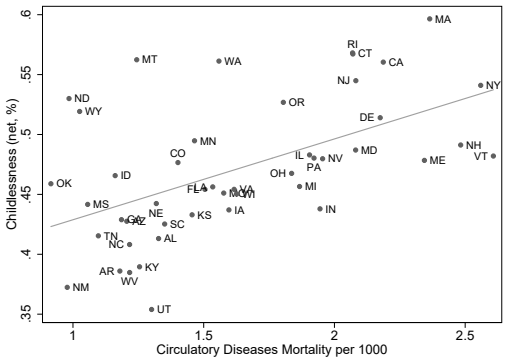
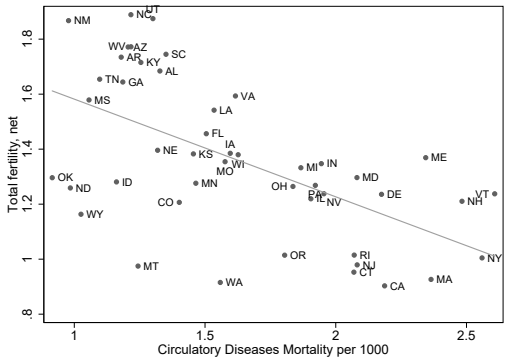


Figure A.8: Mortality from Adult Diseases and Net Fertility in 1930 across US states

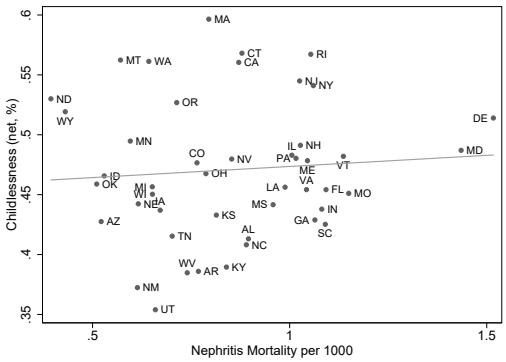
(a) Heart Disease Mortality and Net Childlessness



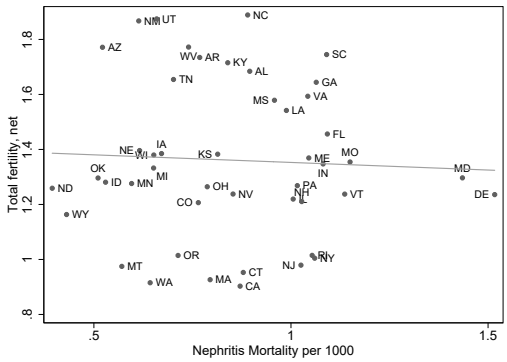
(b) Heart Disease Mortality and Net Fertility



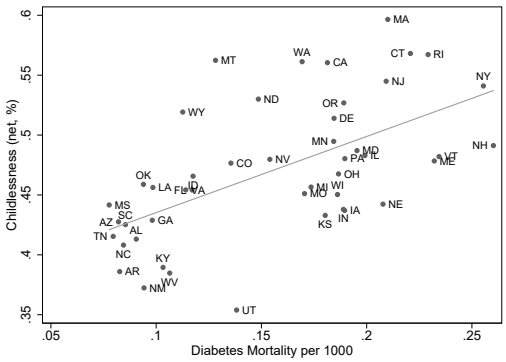
(c) Nephritis Mortality and Net Childlessness



(d) Nephritis Mortality and Net Fertility



(e) Diabetes Mortality and Net Childlessness



(f) Diabetes Mortality and Net Fertility

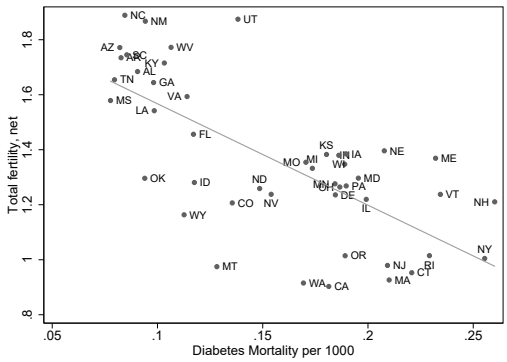
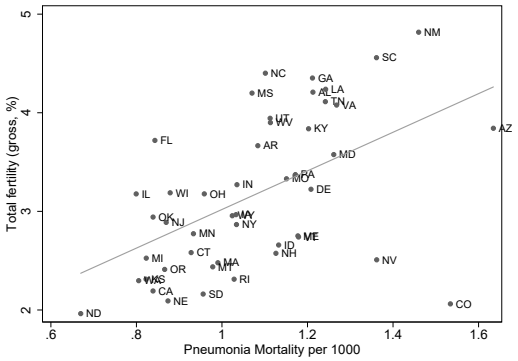
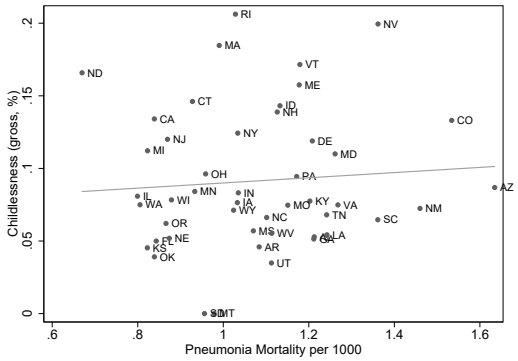


Figure A.9: Cross-Sectional Correlations between Gross Fertility and Child Mortality for 1910

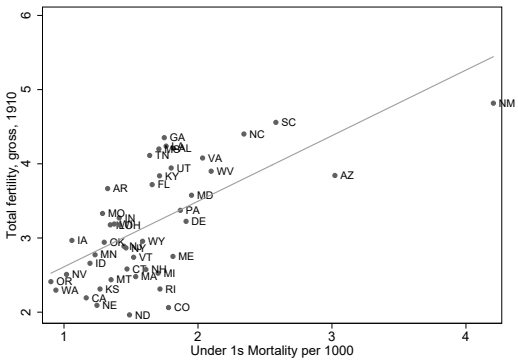
(a) Pneumonia Mortality and Gross Fertility



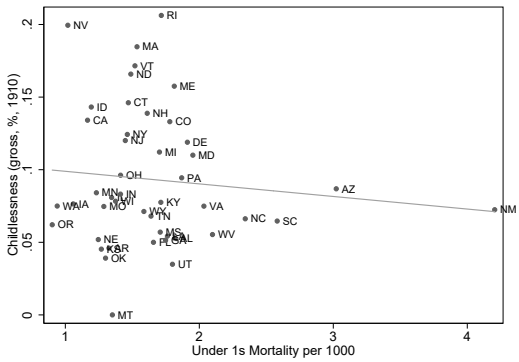
(b) Pneumonia Mortality and Gross Childlessness



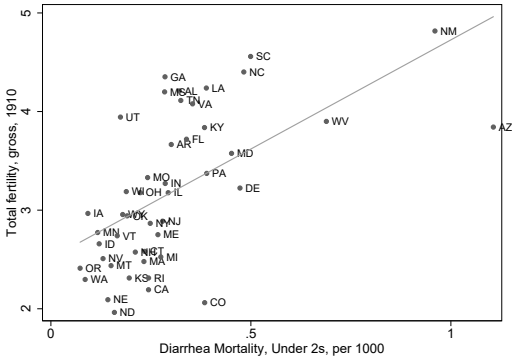
(c) Under 1s Mortality and Gross Fertility



(d) Under 1s Mortality and Gross Childlessness



(e) Under 2s Diarrhea Mortality and Gross Fertility



(f) Under 2s Diarrhea Mortality and Gross Childlessness

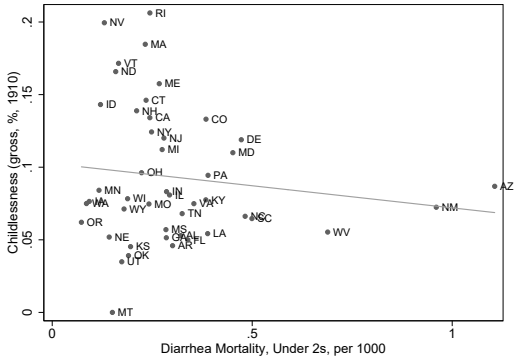
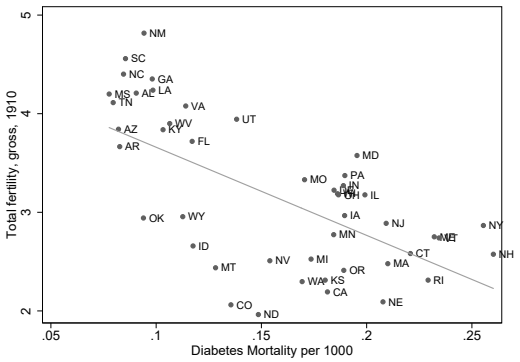
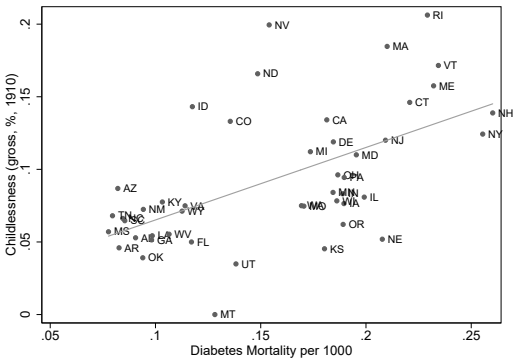


Figure A.10: Cross-Sectional Correlations between Gross Fertility and Adult Mortality for 1910

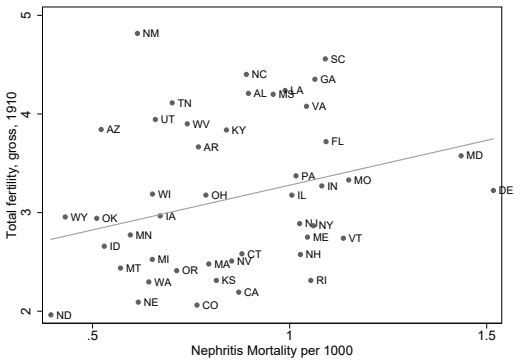
(a) Diabetes Mortality and Gross Fertility



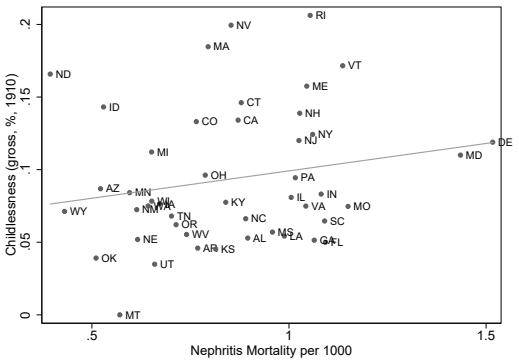
(b) Diabetes Mortality and Gross Childlessness



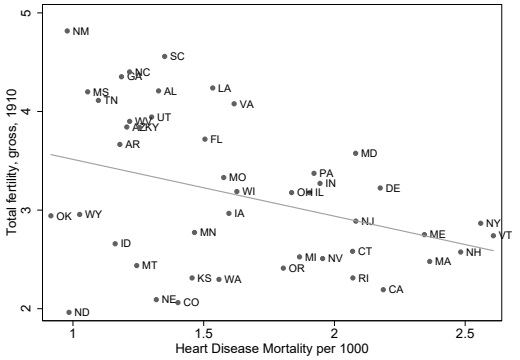
(c) Nephritis Mortality and Gross Fertility



(d) Nephritis Mortality and Gross Childlessness



(e) Heart Disease Mortality and Gross Fertility



(f) Heart Disease Mortality and Gross Childlessness

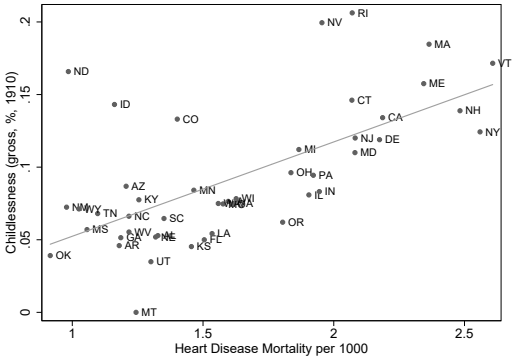
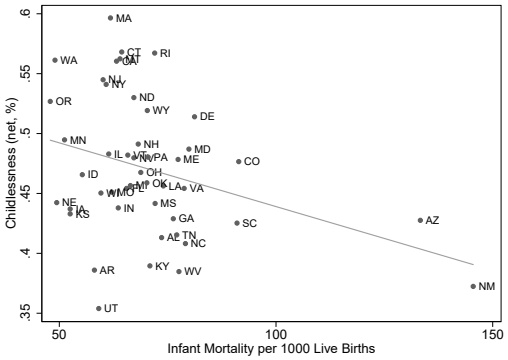


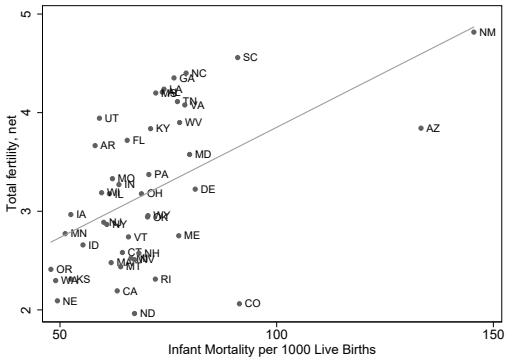
Figure A.11: Cross-Sectional Correlations between Fertility, Labor and Marriage Outcomes and Infant Mortality per 1000 Live Births

(a) Total fertility, net, 1930

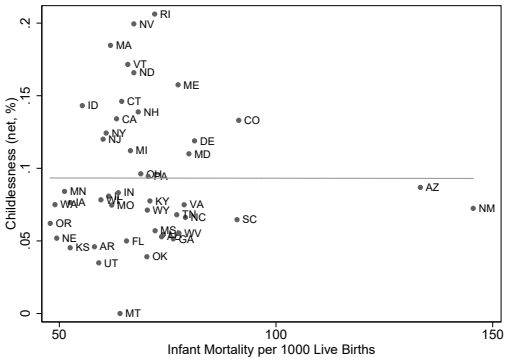
(b) Childlessness, net, 1930



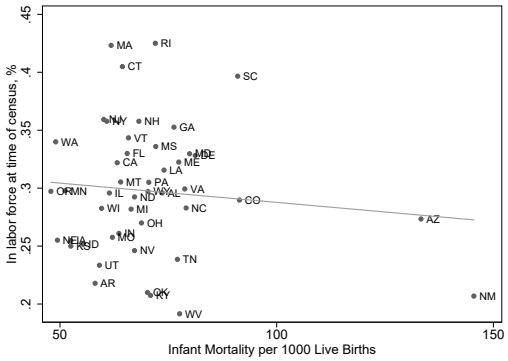
(c) Total fertility, gross, 1910



(d) Childlessness, gross, 1910



(e) Labor Force Status, 1930



(f) Marital Status, 1930

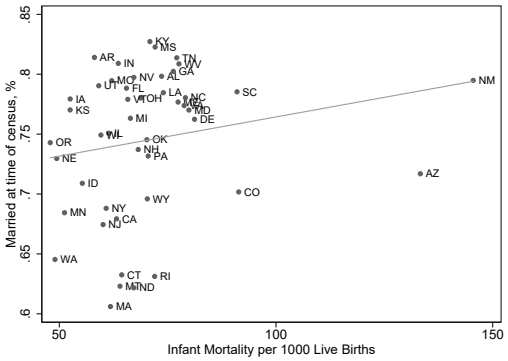
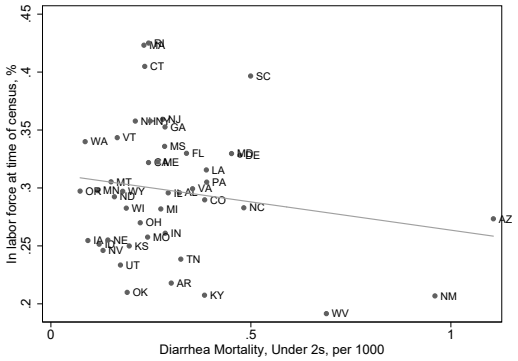
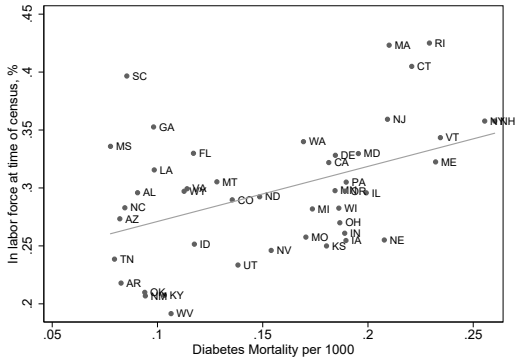


Figure A.12: Cross-Sectional Correlations between Labor Force Status and Mortality Rates, 1930

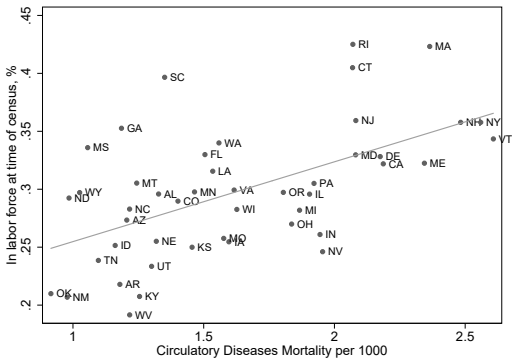
(a) Diarrhea Mortality, Under 2s



(b) Diabetes Mortality



(c) Heart Disease Mortality



(d) Nephritis Mortality

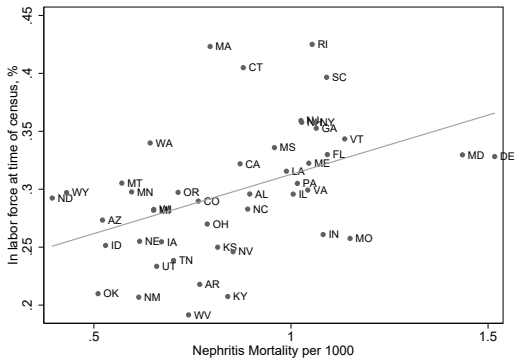
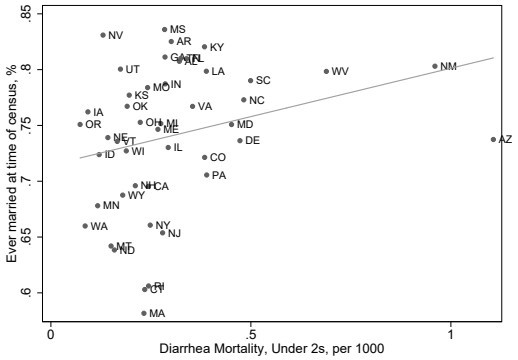
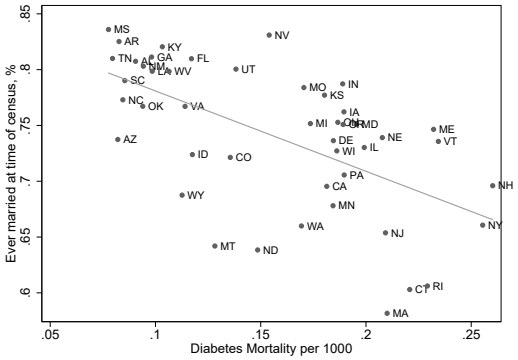


Figure A.13: Cross-Sectional Correlations between Marital Status and Mortality Rates, 1930

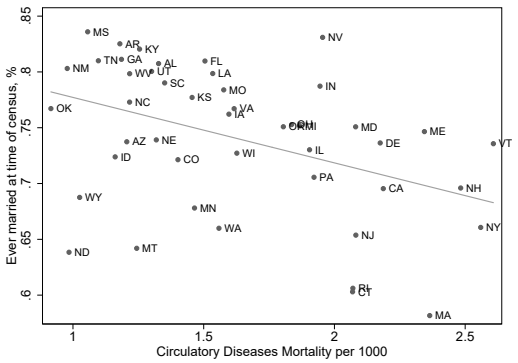
(a) Diarrhea Mortality, Under 2s



(b) Diabetes Mortality



(c) Circulatory Diseases Mortality



(d) Nephritis Mortality